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## REVISION OF THE VENOUS SYSTEM: THE ROLE OF THE VERTEBRAL VEINS.

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The simple believeth every word; but the prudent man looketh well to his going.

—*Proverbs* xiv, 15.

THE discovery of the circulation is perhaps the most fascinating in the history of medicine. It is well for us to reflect on the many stages in the acquirement of knowledge of the circulation over the centuries, for only in this way can we adopt a proper perspective for our study. For it is only when we realize that the conceptions of blood flow put forward by various scholars, anatomists and philosophers throughout the centuries had often lasted several hundred years before being proven false, and that each conception had been invariably proven incorrect, with the exception of Harvey's, that we begin to wonder whether our conception of the circulation today is completely acceptable. As regards the venous part of the circulation, I believe our present conception is incorrect.

### HISTORICAL REVIEW.

For centuries the circulation remained unknown to man. Aristotle (384 to 322 B.C.) believed that the heart contained three cavities. The right cavity contained the hottest blood, the left held the least and the coolest blood. This corresponded to the belief that the right half of the body had a greater temperature than the left. The third cavity was common to both sides.

The theories of Galen (A.D. 131 to 201) were supreme for many centuries. Galen's mistaken ideas were these. Life consisted of three essentials: the first was the "natural spirit", which came to the blood from the liver, the centre of nutrition and metabolism; the second was the "vital spirit", which mixed with the blood in the heart, the centre of the heat regulation of the body; and the third was the "animal spirit", resident in the brain, the centre of sensation and movement. He believed in the anastomosis of arteries and veins and the transfer of the blood and "spirit" from one to the other by very small passages. Galen also assumed communication of the two ventricles by invisible pores in the septum. He realized that arteries contained blood and experimentally proved pulsation in vessels. However, Galen regarded the movement of the blood as an ebb and flow, the arterial blood conveying vital spirits from the heart, the venous blood conveying natural spirits from the liver. Galen also demonstrated the myogenic theory of the heart.

Andreas Vesalius (1514 to 1564), although still ignorant of the presence of the circulation, was outstanding in contradicting many statements of Galen in anatomy in general, including that of the presence of invisible pores in the septum. Leonardo da Vinci probably indicated to his pupils the course of further study.

Until the Renaissance, the liver was regarded as the centre of blood flow. In the liver it was believed that the blood mixed with the chyle which had been brought there by the "meseraic" veins and from there spread throughout the body. The left ventricle of the heart contained air, or blood mixed with air, which reached the right side of the heart through pores in the ventricular septum. The air passing through the arteries carried the vital spirit throughout the body, reaching the heart from the lungs by means of the *arteria venalis*. Only the veins carried blood. Leonardo da Vinci and Vesalius disbelieved the presence of pores in the septum, but not until Harvey were we to get the true conception of circulation.

The discovery of the pulmonary circulation is attributed to Michael Servetus (1509 to 1553). The following is a translation of his work by Willis.

> . . . the first thing to be considered is the substantial generation of the vital spirit—a compound of the

> inspired air with the most subtle portion of the blood. The vital spirit has, therefore, its source in the left ventricle of the heart, the lungs aiding most essentially in its production. It is a fine attenuated spirit, elaborated by the power of heat, of a crimson colour and fiery potency—the lucid vapour as it were of the blood, substantially composed of water, air and fire: for it is engendered, as said, by the mingling of the inspired air with the more subtle portion of the blood which the right ventricle of the heart communicates to the left. This communication, however, does not take place through the septum, partition or midwall of the heart, as commonly believed, but by another admirable contrivance, the blood being transmitted from the pulmonary artery to the pulmonary vein, by a lengthened passage through the lungs, in the course of which it is elaborated and becomes a crimson colour, mingled with the inspired air in this passage . . . and reaches the left ventricle of the heart. . . . The vital spirit . . . is at length transfused from the left ventricle of the heart to the arteries of the body at large, in such a way that the more attenuated portion tends upwards and undergoes further elaboration in the retiform plexus of vessels situated at the base of the brain . . . elaborated by the igneous power of the soul.

It appears that Realdo Colombo of Cremona (1510-?) also observed this independently and unknown to Servetus.

Fabricius (1537 to 1619), although not the first to see them, demonstrated the valves in veins to his pupils, but even so, their real significance was unknown to him.

The conception of the circulation, as we have it, was known to Andrea Cesalpino (1519 to 1603) before Harvey's great work was completed. He was the first to use the word "circulation", and is entitled to a rightful place with Servetus and Harvey. Here is an extract from Cesalpino:

> The orifices of the heart are made by nature in such a way that the blood enters the right ventricle of the heart by the *vena cava*, from which the exit from the heart opens into the lungs. From the lungs there is another entrance into the left ventricle, from which, in turn, opens the orifice of the aorta. Certain membranes placed at the openings of the vessels prevent the blood from returning, so that the movement is constant from the *vena cava* through the heart and through the lungs to the aorta.

Cesalpino altered the centre of the circulation from liver to heart.

In 1628 Harvey's "*De motu cordis*" was published, probably the greatest milestone in medicine. William Harvey (1578 to 1657) appreciated the presence and true significance of the valves; he discovered that they allowed blood to flow only towards the heart. He demonstrated the return of blood to the heart by way of the veins, and also showed that it was a mathematical necessity. He showed that the heart was a muscular pump, and pumped blood into the arteries, from which it flowed to the veins. To quote Harvey:

> It has been shown by reason and experiment that the blood by the beat of the ventricles flows through the lung and heart and is pumped to the whole body. There it passes through the pores in the flesh into the veins through which it returns from the periphery everywhere to the centre, from the smaller veins into the larger ones, finally coming to the *vena cava*, and right auricle. This occurs in such an amount, with such an outflow through the arteries and such a reflux through the veins, that it cannot be supplied by the food consumed. It is also much more than is needed for nutrition. It must therefore be concluded that the blood in the animal body moves around in a circle continuously, and that the action or function of the heart is to accomplish this by pumping. This is the only reason for the motion and beat of the heart.

So much for the general circulation and the pulmonary circulation. It was Malpighi (1628 to 1694) who was first to see the capillaries. To quote him:

> Hence it was clear to the senses that the blood flowed along sinuous vessels and was not poured into spaces, but was always contained within tubules, and that its dispersion is due to the multiple winding of the vessels.

So it was that the link between arteries and veins was uncovered.

In 1664, Willis published his "*Cerebri Anatome*", in which was described the anastomosis of vessels at the base of the brain which now bears his name. Raymond de Vieussens (1641 to 1730) was the first to describe the coronary vessels completely.

Let me quote from Poirier on the portal system.

> *Semblable, selon l'heureuse comparaison de Galien, à un arbre dont les racines plongent dans le tube digestif et la rate, et dont les rameaux s'épandent dans le foie, la veine porte commence dans l'intestin par des réseaux capillaires et finit dans l'organe hépatique par d'autres ramifications analogues.*

I have briefly outlined the history of the circulation, indicating but a few of the famous names associated with the discovery of each system—the general circulation and the peripheral, pulmonary, portal, coronary, and cerebral parts of the circulation.

### THE SCOPE OF THE PRESENT PAPER.

Every reader will be familiar with the present-day conception of the circulation. But do we ever think that it may be erroneous, at least in part? Galen was satisfied with his conception, but nevertheless he was incorrect. Let us now proceed with the discussion of the flow in the venous system, bearing in mind that we may still be short of the truth.

The hub of the discussion centres on Harvey's statement that "the blood in the animal body moves around in a circle continuously". To this we may attribute our modern theory; but it is incorrect, or rather, it is partly incorrect.

Throughout, I shall focus attention upon a great plexus of veins, the vertebral venous plexus, that has escaped the just attention of physiologists and anatomists. This plexus lies within the spinal canal, over the bodies and laminæ of the vertebræ, and partly in the vertebræ themselves. The plexus has been described in the literature, but the writer feels that its true significance has been in great part overlooked. To draw attention to this plexus is all that the writer desires.

For purpose of discussion, the paper is set out as follows: (i) the venous system, as described in current textbooks; (ii) fallacies in this description; (iii) description of the vertebral venous plexus; (iv) physiological and pathological significance of the vertebral plexus; (v) morphology of veins, and the direction of blood flow within them. This paper is a preliminary report, and for the sake of brevity the detailed description of the anastomoses referred to must unfortunately be curtailed. Reference to the work of Poirier and Charpy will supply details of these anastomoses for the interested reader. It may also be mentioned here that experiments are still being carried out, the results of which will be published later. The present paper only presents an hypothesis.

### THE VENOUS SYSTEM AS DESCRIBED IN CURRENT TEXTBOOKS.

According to the descriptions in Gray's and Cunningham's and other textbooks, the veins unite successively until all the blood is returned to the heart by the superior *vena cava* and the inferior *vena cava* (the caval system) and by the pulmonary veins (the pulmonary system). The portal system is regarded as entering into the caval system, and the coronary system opens into the heart directly. The vertebral veins receive scant attention, and although anastomoses are mentioned between the azygos system on the one hand, and the intercostal, lumbar, right bronchial, pericardial, mediastinal and phrenic veins on the other, the significance is not stressed from either the anatomical or the physiological viewpoint.

#### The Fallacies in this Description.

1. The statements by Cunningham that "the superior *vena cava* returns blood from the head, neck, upper limbs, thoracic wall and upper part of the wall of the abdomen", and that "the inferior *vena cava* receives all the blood from the lower limbs, the greater part from the walls and contents of the abdomen and pelvis", are correct only if we imply that the blood destined to return to the heart from those regions must go to the heart by the caval system;

but the presumption that it drains only into this caval system is incorrect.

2. There is a presumption, implied in the above statement, that blood returns almost immediately to the heart via the caval system.

3. There is inadequate description of venous plexuses or "store houses", which are commonly asserted to be in existence; apart from capillary storage pools and the spleen, in common descriptions there is no indication of any venous storage.

4. The vertebral veins, to be afterwards described, are so inadequately treated as almost to be regarded as non-entities.

The above-mentioned so-called "fallacies" apply mostly to modern British textbooks; but in French and German books, as well as in old British books, there are good descriptions of veins and their anastomoses. However, the following exposition of the venous system is not applied, let alone stressed, even though evidence is present.

An attempt will be made to prove the following hypotheses:

1. That venous drainage is not wholly into the caval system, but that by plentiful anastomoses a not insignificant portion goes into the vertebral veins from head, neck and limbs, thoraco-abdominal walls and pelvis; and also, that various viscera are directly drained into the vertebral veins; the significance of this will be seen later.

2. That the venous system is much greater in volume than the arterial system, for often two veins occur with each artery, veins occur in regions where there are no arteries, and veins are on the whole larger than arteries. Therefore, all the blood may not necessarily go straight back to the heart to maintain the circulation.

3. That the vertebral system is a very large system and contains a great volume of blood exceeding that of most venous stores. An amount of 200 millilitres of dye was found by Batson to be comfortably accommodated in it, even though the cadaver was small. Besides storage, this system anastomoses with all other parts of the circulatory system, except the coronary circulation, and it is a means of regulation of the cerebro-spinal fluid pressure.

I shall now discuss points in the return of blood from (*a*) the head, (*b*) the pelvis, and (*c*) the body walls.

### *The Head.*

The following data indicate that the statement that all the blood returns from the head and neck to the superior *vena cava* is not absolutely correct. Professor Shellshear has thrown some light on the subject. He points out that in our own day cadavera are preserved in formalin, which produces hardening and contraction of the veins, and this is partly the reason why advances in the description of veins have not occurred since Quain's time in the middle of the last century. He quotes from Hilton:

> There is a tendency which is not only prevalent amongst students but even amongst others, to ascribe to the internal jugular veins a more important part than they really perform, or to attribute to them a greater share in the return of venous blood from the brain than they really take, and to regard in a less important light than they really deserve those accessory systems which escape in various points through the osseous walls of the skull. Not only do these accessory systems convey from the brain a considerable portion of blood under the normal, but also under the abnormal conditions of life. For when there exists a temporary venous obstruction in the lungs and heart, they contribute the chief and almost only means of escape of venous blood from within the cranial cavity. If, for example, from a voluntary effort or from some other cause, the process of respiration be arrested, we know as a matter of observation, the eyes start and that the face becomes exceedingly red and turgid.

This paragraph appears to have been left unnoticed by physiologists.

Hilton realized the value of accessory pathways, especially in obstruction of the internal jugular vein. The most well known of these accessory pathways are grouped together as emissary veins; but as the function and importance of these are so well understood, they will not be described in detail. Rather, attention will be drawn to another accessory pathway, less well known, but probably of much greater physiological importance. This accessory pathway is the vertebral venous system. The superior extremity of the internal vertebral venous plexus lies on the clivus of the skull around the margins of the *foramen magnum* and in the region of the atlas. Thus, at the base of the skull, this plexus anastomoses with the great venous trunks of the cranium, and is therefore admirably situated to perform its role as an accessory pathway. From its commencement on the inside of the base of the skull, and throughout its whole length, this internal vertebral plexus is encased in bone and is therefore free of the effects of external pressure. In the neck, the vertebral veins make anastomoses with the deep cervical veins.

In the presence of obstruction to the internal jugular vein, this accessory pathway will probably exceed in importance the pathways via the emissary veins, for the latter only change the position of the blood within the same venous drainage area (that of the internal jugular), whereas the former carries the blood out of the drainage area of the internal jugular into a new drainage area, the vertebral system. This change of venous drainage area provides a real escape for the blood dammed up in the internal jugular drainage area.

In other words, the internal vertebral plexus provides the only true alternative pathway of venous return from the cranium, and therefore overshadows the emissary venous system in importance. In fact the writer believes that the emissary veins have been credited with too much, and have gained a distinction that more truly belongs to the vertebral veins. For on comparing the size of the two pathways and the situation of these two alternative routes, and on considering the problem of drainage areas (see above), one is forced to believe that the emissary veins have been credited with much more than they really deserve, and what is more, they have been credited with a function that is largely carried out by the vertebral veins, and only to a lesser extent by them. It is the eclipse of the vertebral veins in modern descriptions that is to blame. Therefore, the writer believes, we are correct in regarding the vertebral veins as the largest and by far the most important accessory pathway for venous return from the cranium.

Morphologically, the cranial sinuses and internal vertebral plexus are in the one plane; but at the base of the skull the venous blood in the sinuses gains the plane of the "active stratum" or plane of the viscera, and is carried directly to the heart via the internal jugular vein. Thus, although the blood does not normally continue in the same morphological plane, yet, in the presence of obstruction to flow in the internal jugular vein, it is only natural that the flow will be diverted into the vertebral veins which are in the same morphological plane as the sinuses. Thus, morphologically, the internal vertebral plexus is the ideal alternative pathway.

Also we must remember that the emissary foramina decrease in size with age; in other words, the importance of the emissary system decreases with age.

Thrombosis of the superior *vena cava* does not mean loss of life from venous obstruction in the head and neck. The operation of ligation of the internal jugular vein in transverse sinus thrombosis does not mean venous obstruction in the head. The Queckenstedt test would not be so freely applied in wards if obstruction of the internal jugular vein was so serious. Thus the following statement of Morris is not correct:

> All veins, whether superficial or deep, sooner or later terminate in the internal jugular, external jugular, vertebral or deep cervical—chiefly the two former, and these veins open directly or indirectly into the innominate veins in the root of the neck through which all the blood from the head and neck passes to the heart.

There appears to be another pathway for the drainage of the blood of the head, and this functions to great effect in caval obstruction. This pathway is the vertebral veins.

*The Pelvis.*

The following observations disprove the statement that all the blood returns directly from the lower part of the body to the inferior *vena cava*. Batson approaches the subject from the viewpoint of cancer spread, being dissatisfied with the view that spread of prostatic cancer is by way of lymphatics. He injected cadavera, using Weber's "king's yellow" (an artists' tube water colour), which is radio-opaque and is readily followed by fluoroscopy. When the dye was injected into the dorsal vein of the penis, which is virtually the same as the prostatic plexus, it was noticed that it went partly into the inferior *vena cava* and partly into the vertebral veins. But when a dye especially for fine vessels was used (Weber's artists' water colour vermilion, because it casts a good shadow in small dilutions), it was found that the dye did not go into the caval system, but into the pelvic veins, the sacral body and the wings of the ilia (vertebral veins). This illustrates remarkedly well the distribution of spread from prostatic cancer which is found clinically, and it is noted that the distribution does not correspond with the lymph drainage pattern. On injection, the dye travelled up the epidural and vertebral veins, freely anastomosing with those of the thoraco-abdominal cavity to the skull. Even when 200 millilitres of injected fluid were used, the dye did not go into the caval system. When live monkeys were used (the experiment was repeated under anæsthesia), the dye found its way into the inferior *vena cava*, but when a towel was placed around the abdomen, the flow was into the vertebral veins. This shows that the valveless and low pressure system of vertebral veins is important. The caval system is the usual way of return during activity, but the vertebral system is the way when pressure is increased in any cavity. Injection into the vein of the breast of a monkey shows that the dye travels to the clavicle, the intercostal veins, the head of the humerus and the cervical vertebræ. It even finds its way into the transverse sinus and superior longitudinal sinus.

Batson continues: "There is a pathway up and down the spine which does not involve the heart and lungs and it has many connections. It provides a vehicle for tumor metastases and removes the stumbling block of the lung capillary bed." He considers that there are four systems of veins: (i) pulmonary, (ii) caval, (iii) portal, (iv) vertebral—with significant physiological and pathological aspects.

In coughing, in straining, and in Valsalva's experiment, blood is not only prevented from returning to the lungs, but is squeezed into the vertebral vein system. Under the subheading "Vertebral Veins and Their Connections", Batson continues to the effect that they are thin-walled and when empty of blood are barely recognizable. There are rich anastomoses in the head (the brain, the meninges, and the bone of the skull, which are storage places for blood, and are a cause of stagnation). Batson considers the vertebral system as a lake or blood store. The longitudinal veins, he points out, are duplicated in size and pattern from segment to segment, and have connexions with veins of body cavities at each intervertebral space. They also have rich connexions with the veins of the spinal column and spinal cord. The system communicates with segmental veins—for example, the intercostals, those of the breast and the azygos veins—and via these, it communicates with pleural and bronchial veins. Rich connexions occur with pelvic viscera, and occasionally it communicates with the renal veins. In a male cadaver only five feet four inches in length and weighing 65 pounds (a small person), 200 millilitres of medium were put into the vertebral venous system. This illustrates its capacity. The testes and ovaries have no connexion with the system. Batson states that pelvic veins have varying valves. Veins accompanying spinal nerves are described as having valves, but are known to be no resistant barrier.

This probably explains the occurrence of metastases in the brain in cases of lung cancer (or abscess). Cancer causes cough, this causes increased intrathoracic pressure, blood from the bronchial veins is squeezed into the vertebral system, whence it is carried into the brain. Batson concludes by stating that it is a venous pool and also a "by-pass". His statement is accompanied by a diagram illustrating his "fourth" system.

*The Body Wall.*

From the foregoing observations, and from our knowledge of anatomical anastomoses, we know that the intercostal and lumbar veins connect with this vertebral system as well as with the caval system. By the arrangement of the valves in the posterior segment of the intercostal veins, it seems that the direction of flow is back into the azygos and vertebral system.

**Comment.**

Therefore, we see that it is a fact beyond doubt that the blood from the head, pelvis and body wall can and does flow, in part, into the vertebral veins. Even though a degree of obstruction in the caval system is required to cause this flow to occur to any extent, it is immensely important to realize that these obstructions are being caused every few minutes of our lives from coughing, sneezing, straining, micturition, defæcation, parturition *et cetera*. That is, there is a steady but intermittent flow into and out of the vertebral veins.

DESCRIPTION OF THE VERTEBRAL VENOUS PLEXUS.

With an idea of the importance of the vertebral system, it is now possible to describe the anatomy of the veins themselves, and it will be found that the description fits

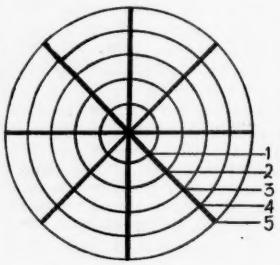


FIGURE I.

Diagram representing the basic architectural design of the arterial system (shown as radii radiating from a central heart) and venous system (shown as concentric circles). The venous strata are: (i) cerebro-spinal stratum, (ii) epidural stratum (internal vertebral plexus), (iii) bony stratum (diploic veins), (iv) "active" stratum, (v) subcutaneous stratum. The diagram is of a horizontal section of the body.

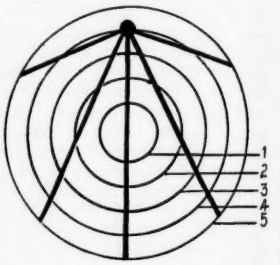


FIGURE II.

Diagram illustrating a more correct interpretation of the basic architectural design of the arterial and venous system. The heart is shown in its correct stratum, the fourth, and not in the centre. The venous strata are: (i) cerebro-spinal stratum, (ii) epidural stratum (internal vertebral plexus), (iii) bony stratum (diploic veins), (iv) "active" stratum, (v) subcutaneous stratum. The diagram is of a horizontal section of the body.

in well with what we have now formulated in our mind. (*a*) It is a "store-house" of blood, and this we see in the architecture of its dilatations and in its very magnitude. (*b*) It is a pool for receiving "backflow" from adjacent veins, hence its many anastomoses. (*c*) It redistributes the blood from other adjacent parts, and the backflow from other regions is soon accommodated in its very immensity; it reminds us of the "invaders" of China, who are absorbed until they themselves become Chinese. (*d*) Any unequal pressure in the adjacent veins is quickly equalized, and this follows as a result of the greatness of the system's extent, its low pressure, the shortness of its component vessels, and the dilatations in each segment. (*e*) It itself has no pressure, and hence is more suitable to act as a pressure absorber. (*f*) It has no direction of flow, and this makes possible a quick adjustment and accommodation to a sudden inrush of blood; this is shown in its consisting of a network instead of longitudinal channels.

The following statement is from the works of John Hunter:

> The vessels carrying blood from any part of the body to the heart are called veins. They are more passive

than arteries and seem to be from their beginning to their termination in the heart little more than conductors of blood to the heart, that it may receive its salutary influence from the lungs. However, this is not universally the case, for the *Vena Portæ* would seem to assume the office of an artery to the liver and therefore an active part: and we have many veins formed into plexuses so as to answer some purpose not at all subservient to the circulation, but still in this respect they are not to be reckoned active. They differ from the arteries in many of their properties, although in some ways they are very similar. They do not compose so uniform or regular a system of vessels as the arteries either in their form or use, being subject to considerable variety in their uses (which are, however, passive and not active) and often answering from their construction collateral purposes.

This last sentence shows that Hunter thought that veins were built for many purposes and that their structure was in conformity with their function.

We have the following clear description of these veins in Quain:

The veins which return the blood from the spine and parts contained within the cavity, present some peculiarities which distinguish them from those in other situations. Proceeding from the posterior surface of the spine to the anterior aspect, we find first a series of tortuous veins deeply placed in the vertebral grooves between the extensor muscles; in the next place a complete network of veins surrounds the spinal canal on its interior surface and two venous reservoirs extend along its entire length not resembling sinuses (for they are not formed in *dura mater*) nor ordinary veins, for they do not present a continuous canal: they resemble rather a chain of short veins linked together receiving blood from the other rachidian veins and transmitting it to some part of the general venous system. The great spinal veins lie along the interval at each side between the bodies of the vertebra and the intervertebral foramina. In some parts, the links of the chain they form are double or even triple, and occasionally detached from any connection with the link above or that below which shows that each portion is, as it were, a separate trunk itself receiving the blood on the one hand and propelling it on the other hand, and that it does not therefore ascend or descend along the column which the series of veins form. Each of these venous links is as long as the interval between two intervertebral foramina: for it is found constricted at both extremities which communicate by short narrow canals with the veins at the forepart of the spine. In the thoracic region they open into the azygos and into the intercostal veins; in the neck for the most part into the vertebrals. A complex interlacing of tortuous veins is established along the inner surface of the arches of the vertebræ. In the lower part of the canal the interlacement is not so close as in the upper portion where it usually conceals (if the injection has run minutely) the whole surface of the *dura mater*. These veins also converge to the intervertebral foramina and open by rather narrow channels into the intercostal veins. The numerous foramina observable in the bodies of the vertebra find exit to veins which open into the great spinal veins. Another group of veins—*venæ dorsispinales*—arise amongst the extensor muscles and pass in a tortuous course forwards to reach the spaces between the arches of the vertebræ and open into the mesh of the spinal veins after having pierced the *ligamenta sub-flava*. Some, however, accompany the posterior branches of lumbar and intercostal arteries and pass through the spaces between the transverse processes of the vertebræ, to open into lumbar or intercostal veins. These veins literally encircle the root of the transverse process . . . in other parts of the venous system, the blood flows in vessels, forming continuous tubes which diminish in number, as they increase in size, in their progress forwards towards the heart, each tributary current going in most instances at an acute angle with the larger one into which it flows: but here the blood from the muscles and the interior of the spine is conveyed into the great spinal veins which are wider in the middle than at their extremities and therefore resemble so many reservoirs from which it flows off by outlets or minor veins terminating in the general venous system. What is the propelling force? . . . M. Breschet conceives that the circulation in the great rachidian veins presents several points of similitude with that in some animals that occupy the lower grades in the scale of being and that it is performed with a degree of slowness, proportionate to the impediments of its course.

The anastomoses of this vertebral venous system are legion, and they have been described by Poirier and Charpy and by many other writers. Although space does not allow me to enumerate all these anastomoses, yet I must point them out in a general manner.

The superior termination of the internal vertebral plexus lies, as already described, on the clivus of the skull and in the region of the *foramen magnum*. Thus it anastomoses with the other venous channels inside the skull. In the neck there are anastomoses with the deep cervical veins, and in the thorax and abdomen the anastomoses with the intercostal and lumbar veins are well known. Inferiorly, the internal vertebral plexus has communication with the large pelvic plexuses, by vessels passing through the anterior sacral foramina; and also, by channels passing backwards through the posterior sacral foramina, anastomoses occur with the gluteal veins. Extending almost the length of the trunk, the vertebral system is able to anastomose with veins in the superior and inferior poles of the body, and with the veins on both sides of the trunk; so advantageously situated is this plexus that it anastomoses with all portions of the venous system, excepting the coronary circulation, and it is therefore able to exert a profound influence on venous flow and venous pressure, for in the first instance, it provides an alternative route for venous blood, and in the second, it has a presumably lower pressure than the caval system.

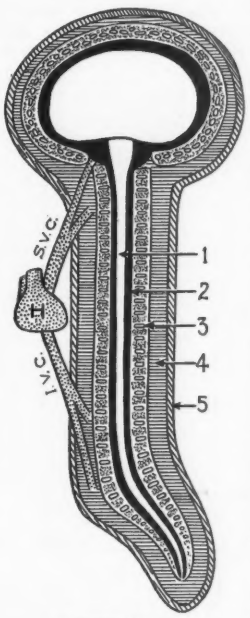


FIGURE III.

Diagram illustrating the various venous strata of the body; (i) cerebro-spinal stratum, (ii) epidural stratum (cranial sinuses, internal vertebral plexus), (iii) bony stratum (diploic veins of the skull, intravertebral veins), (iv) "active" stratum (plane of soft tissues, viscera, muscles *et cetera*), (v) subcutaneous stratum. H—heart; S.V.C., I.V.C.—superior and inferior *vena cava* respectively. The diagram represents a longitudinal section of the body.

But these are not the only anastomoses of this vertebral system, for various viscera—for example, the lungs, the left suprarenal and the left kidney—have a direct anastomosis with the vertebral veins. These communications are listed below and are of great pathological importance, for at all times a great portion of the venous blood from these organs flows into the azygos and vertebral veins, whereas in the case of most other organs—for example, the pelvic viscera—a change in thoraco-abdominal pressure is necessary to direct the venous flow into the vertebral system.

### Bronchial Veins.

There are two bronchial veins on each side, which are not quite satellites of the bronchial arteries, and which not only drain the small and larger bronchi, but also receive the drainage of the lymph nodes of the hilum, which is of great importance in cancer spread. The right empties into the arch of the azygos, the left into the superior hemi-azygos.

### The Inferior Hemi-Azygos.

The inferior hemi-azygos commences by the union of the left ascending lumbar vein and an anastomosis with the left renal vein and enters the *vena azygos*. It often communicates with the spermatic and left suprarenal veins (see below, Hutchison's syndrome). We must remember

that the azygos veins are intimately related to the vertebral plexuses.

### Anastomoses with the Portal Vein.

The œsophageal and phrenic veins drain into the azygos system, and the system of Retzius allows anastomosis with the vessels of the posterior abdominal wall and with the veins around the spinal column.

### The "Reno-Azygo-Lombaire" Anastomosis.

The "*reno-azygo-lombaire*" anastomosis of Lejars, according to Testut, opens by one end into the renal vein, and after bifurcating near its other end, empties into the inferior hemi-azygos and first lumbar vein. Testut states that it is present in 88% of cases, and Poirier and Charpy found it present in 62 of 70 cases on the left side and in six only on the right side. On the right it is usually replaced by an anastomosis between the renal vein and first lumbar vein. Therefore, this vital renal anastomosis is usually on the left side.

### Comment.

By this time we have now a clear conception of the vertebral veins. A glance at the vertebral veins in the atlas of Toldt gives us a striking illustration. They are a distinct system, warranting every bit of Batson's enthusiasm—they have wide anastomoses with the rest of the veins of the trunk, yet the whole system is one that deserves recognition as an entity. Apart from being an anatomical fact, it will now be shown that it is a system of physiological and pathological importance, as it holds within itself many secrets of as yet unknown physiological phenomena. It is with this that I shall now deal. From its widespread anastomoses it may explain some of those facts of cancer spread of which the explanation is as yet so far from our grasp. As a surgical problem, its very massiveness is of interest in spinal surgery, not only in the attack on the spine itself, but in the posturing of the patient, as it has long been known that hæmorrhage occurs more readily when the patient is prone (compression of abdomen).

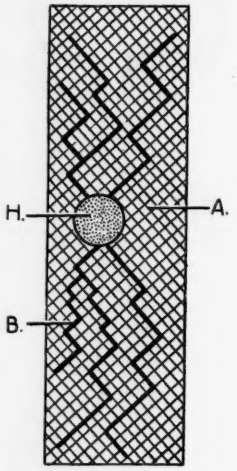


FIGURE IV.
Diagram illustrating the development of channels (B) in the venous plexus (A). H, heart.

### The Physiological and Pathological Significance of the Vertebral Venous Plexus.

The relation of the vertebral venous plexus to cerebro-spinal fluid pressure illustrates well the physiology of the veins themselves. Thus, this section will deal at length with the problem of maintenance of and of variations in the cerebro-spinal fluid pressure, as far as it throws light on the plexus under discussion. As I have no experimental evidence to offer as yet, and as my work is put forward only as an hypothesis, no attempt is made to discredit theories based on experimental fact.

Hamilton, Woodbury and Harper have written an article on the physiological relationships between intrathoracic pressure, intraspinal pressure and arterial pressure. They make the following statement:

> Evidence to be presented elsewhere shows conclusively that quick changes in intrathoracic pressure are transmitted directly and immediately to the cranio-spinal canal. These pressure changes are NOT propagated to the c.s.f. by the internal jugular vein, because they are slower and less extensive in that vessel than in the canal itself. They are NOT propagated over the arterial tree. An ordinary systemic rise of arterial pressure causes a rise of intraspinal pressure of only 1-2 mm. Hg. A similar rise in arterial pressure caused by a cough is accompanied by a rise in intraspinal pressure as great or even a bit greater than the arterial rise itself. This evidence leads us to regard the cranio-spinal cavity as a sort of functional extension of the thoracic cavity and to think of the cerebrospinal arteries as protected from the sudden stress that results from straining and coughing in the same fashion that the intrathoracic arteries are protected. Thus in Fig. 5 [of these authors' work] the rises in systemic B.P. that occur as a result of coughing do not appear in the nett intraspinal pressure. They are cancelled by similar rises in c.s.f. pressure. The nett intracranial B.P. fails to rise as does the systemic B.P., but just as in the thorax, it does rise after strain is over. Moreover, the intracranial pressure pulsations are much more extensive after a strain puts the greatest stress on the cerebral arteries. The nett intraspinal B.P. which is analogous to the nett intrathoracic B.P. is, of course, the force that bursts the arteries and causes apoplexy. One cannot fail to be struck by the admirable way in which the vital arteries within the cranio-spinal, within the thoracic, and to a great extent within the abdominal cavities are protected against these sudden and enormous physiologic increases in pressure. Admitting the existence of these pressure relationships and their usefulness, what is there to say about the mechanism that results in the prompt and opportune rise in intracranial pressure? As we have seen, the pressure cannot be propagated from the thoracic to the craniospinal canal by the arteries or by the internal jugular vein. How then do these sudden and large pressure changes occur in such a rigid box as the craniospinal canal? These are the hypotheses. We would suggest that the pressures which arise during coughing and straining in the thoracic and abdominal cavities are transmitted through the soft tissues between these cavities into the spinal foramina. Because of the rigidity of the cerebrospinal canal, and because of the temporarily increased vascular pressure no very large tissue or fluid volume would need to be squeezed into the canal in order to raise the pressure even to the extent it does during cough. The material entering the canal under stress may be: (1) C.S.F. from under evaginations of the dura around spinal nerves. (2) Spinal nerves themselves and the loose areolar tissue surrounding them. (3) Venous blood in the para-vertebral plexus, and in the veins at the back of the thorax and abdomen in close communication with the veins. . . . The pressure increases that are, according to our notion, produced in the spinal canal must naturally be propagated up through the *foramen magnum* and into the cranial cavity. Even though the volume movement up through the base is small, the pressure thrust is considerable. A similar and opposite thrust occurs as intraspinal pressure goes down. The fact that these stresses are localised at the base of the brain may have some bearing on the frequency of hæmorrhage in this region.

With that, Hamilton, Woodbury and Harper concluded their paper.

From this I developed the notion that in increased intrathoracic pressure, it was most likely a squeezing of the blood vessels in the thoracic walls that increased the volume of the vertebral veins. How, then, was this transmitted to the cerebro-spinal fluid? It would not be surprising if the internal vertebral plexus is larger than what we expect. In the days when alcohol and not formalin was used for embalming, the veins were found in a better state, and their size struck the earlier anatomists. It is not far from the imagination, when we consider that in every part of the body no gaps are left between adjacent tissues, if we postulate that most probably the veins within the spinal canal nicely fill up the space in the canal left by the spinal cord and membranes. That is, a mutual pressure may exist between the venous pressure in the veins and the cerebro-spinal fluid pressure. Rise in pressure in the one means displacement of fluid in the other to allow accommodation (however temporary) of the increased quantity of fluid in the one in question.

Clinically, we may take the Queckenstedt test as an example. Pressure on the internal jugular vein causes an immediate rise of cerebro-spinal fluid pressure, and release of the hand causes almost as sudden a fall. Secretion of cerebro-spinal fluid due to the back pressure in the internal jugular vein is no longer feasible as the cause of raised pressure. Events are too rapid for this. From what has been said before, pressure on one internal jugular vein causes the opening up of anastomoses, most likely the vertebral

veins. This swelling of the vertebral veins causes mutual pressure on the subarachnoid space with consequent rise of cerebro-spinal fluid pressure. Release of the hand allows blood to flow quickly onwards in the internal jugular vein, and the stream into the spinal canal diminishes and thus cerebro-spinal fluid pressure becomes normal.

In Froin's syndrome the block in the subarachnoid space is itself of such a nature as to exert pressure on the spinal veins, so that in performing the Queckenstedt reaction we have a column of blood running down only to the site of the lesion—the collapse of the veins at this site prevents the flow going down to the region of the lumbar puncture needle. Thus there is no alteration in this region of the "mutual pressure" relationship, and the column of cerebro-spinal fluid does not rise. It may be more accurate to postulate that it is not the increased flow that is prevented from passing the site of the lesion, but probably the tumour in the subarachnoid space prevents the distribution of the rise in cerebro-spinal fluid pressure above the lesion to the region below. Thus the response to the Queckenstedt test is "negative".

In point of fact, the supposition of this "mutual pressure relationship" is borne out by the observations of Scott and Furlow. These authors describe a new technique for the removal of "Pantopaque" in the spinal subarachnoid space. With the lumbar puncture needle *in situ*, the patient performs the Valsalva manœuvre, which raises intraspinal pressure.

> During Valsalva's manœuvre, two things happen to the column of opaque oil: first, the whole column tends to move craniad a distance of from 0·5 cm. to as much as 5-8 cm.; second, the column becomes narrowed. These changes are the result of engorgement of the venous plexus that surround the dura and can be demonstrated by taking spot films.

During the raised pressure, the lighter cerebro-spinal fluid is displaced and the oil is in more mutual contact with the needle and can be expelled.

Therefore, from the evidence at hand, we see that in such drastic exercises as Valsalva's manœuvre an actual blood shift occurs into the vertebral veins, and this shift of blood with consequent engorgement of the plexus causes compression of the spinal subarachnoid space and raised cerebro-spinal fluid pressure. Thus it appears that in such drastic manœuvres the raised intrathoracic and intra-abdominal pressures are responsible for raised cerebro-spinal fluid pressure.

But what of the effects of the constant, normal respiratory variations on cerebro-spinal fluid pressure? I refer the reader to O'Connell's work. This writer sets forth the accepted views on cerebro-spinal fluid pressure and cerebro-spinal fluid circulation. These are as follows, as O'Connell has set them out:

1. The normal positive intracranial pressure is due to the balance existing between the process of production and absorption of cerebro-spinal fluid.

2. Most observers believe that during a given time of observation, the cerebro-spinal fluid pressure is relatively constant and subject to but slight and unimportant variations with cardiac and respiratory activity. Smith and Kubie (according to O'Connell) point out that the pressure variations with cardiac activity amount to two to four millimetres of cerebro-spinal fluid, and with respiratory activity to five to ten millimetres of cerebro-spinal fluid at lumbar puncture. Frenfield and Carmichael (according to O'Connell) point to the waves superimposed by this activity upon the tide of the circulation between the points of production and absorption. They believe that the replacement of fluid in the spinal canal is due to the pulsations of cardiac and respiratory activity.

3. The circulation of cerebro-spinal fluid is accepted as being a movement from its point of production towards the villi; also the circulation is believed to be slow.

O'Connell disagrees in great part with the above statements. Let us consider each in turn.

1. O'Connell came to the conclusion that vascular factors were of great importance in the maintenance of cerebro-spinal fluid pressure. For he found, in cadavera obtained soon after death, that on lumbar puncture no fluid dripped from the needle, and no manometric record was possible. Rightly he asks why the cerebro-spinal fluid pressure drops to atmospheric pressure after death. With death the production and absorption of cerebro-spinal fluid cease, so that the fall in pressure must be due to increase in volume of the subarachnoid space. O'Connell attributes this to post-mortem decrease in volume of the arterial tree, and to collapse of the intracranial veins when blood fails to reach them from the arteries. Thus vascular factors are important, according to him, in the maintenance of cerebro-spinal fluid pressure.

We have seen the effect of engorgement of the vertebral veins on the cerebro-spinal fluid pressure, and when we compare the size of these to the size of the intracranial veins, it is more probable that the collapse of the former will contribute more to the enlargement of the subarachnoid space than the collapse of the latter. So it may be feasible to state that the vertebral plexus plays a major role in the maintenance of cerebro-spinal fluid pressure.

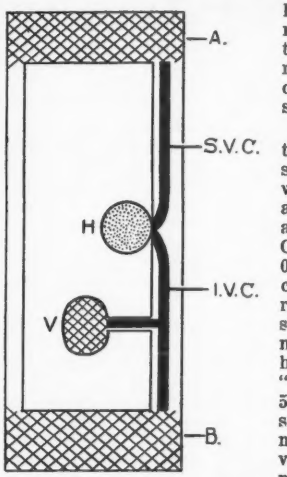


FIGURE V.

Diagram illustrating the effect of the development of the thoraco-abdominal cavity on the production of great vessels and obliteration of the plexus in the area of its development. The poles of the body, into which the cavity does not extend, remain plexiform and are shown as the superior (A) and inferior (B) polar plexuses. The great vessels (S.V.C., I.V.C.—superior and inferior *vena cava* respectively) and heart (H) are shown. The plexuses of the viscera remain despite the body cavity (V).

2. When the subject is in the horizontal position, only small variations are found with cardiac and respiratory activity at lumbar puncture and cisternal puncture. Cardiac variations are usually 0·5 to 1·0 millimetre of cerebro-spinal fluid, and respiratory variations are slower and amount to two millimetres. But, on the other hand, in intraventricular "tap", cardiac variations are 5 to 50 millimetres of cerebro-spinal fluid (average 15 millimetres) and respiratory variations are 15 to 60 millimetres (average 35 millimetres). Thus, according to O'Connell, intraventricular pressures are far from constant and range between 20 and 110 millimetres of cerebro-spinal fluid (average 50 millimetres). O'Connell sought the reason for this difference. By experiment, he has shown that the difference in the readings is due to the difference in bore of the lumbar puncture needle and intraventricular cannula. The former are much narrower in bore, and this smallness of bore prevents the recording of rapid changes in pressure, and even when the changes are recorded, the amplitude is recorded at a much smaller figure than what it really is. Therefore the intraventricular cannula gives the true result. Thus cardiac and respiratory activity, contrary to previous opinion, causes great and important changes in cerebro-spinal fluid pressure. When the subject is in the horizontal position, according to O'Connell, cerebro-spinal fluid pressure varies rhythmically every three or four seconds through a range of eighty millimetres. This accounts for the pulsation of the cerebrum at operations, and for the jet of cerebro-spinal fluid that leaves the cannula periodically in cranial operations.

Backflow into the vertebral veins with respiratory activity is therefore probably considerable, and it is no doubt owing to the engorgement of this plexus and its collapse with respiratory activity that we have this great variation in cerebro-spinal fluid pressure. Other factors associated with cardiac and respiratory activity, as suggested by O'Connell, play their part. But in view of the amount of blood flowing back into the plexus, and of its much more intimate relation with the thorax when compared with the cerebral veins, it must be recognized that

the vertebral plexus is probably the chief factor in these variations.

O'Connell gives us another interesting example of cerebro-spinal fluid mechanics. If a rigid cylinder full of fluid is connected to a manometer and its level is varied, no variation in fluid level occurs. If now the cylinder is provided with a vent, open to atmospheric pressure, then alteration in fluid level occurs with similar alteration of the level of the cylinder, and is recorded on the manometer. In lumbar puncture, if the head is lowered out of the horizontal, the cerebro-spinal fluid pressure falls; but if the head is raised or the sitting posture is assumed, the cerebro-spinal fluid pressure rises. That is, the subarachnoid space is "vented". O'Connell believes that in the raising of the head (or in sitting), cerebro-spinal fluid passes into the spinal theca, raising the pressure there, and at the same time, venous blood is retained in the intracranial cavity to replace the cerebro-spinal fluid. The reverse occurs on lowering of the head, the cerebro-spinal fluid being replaced by an influx into the extradural veins (O'Connell). Thus, the so-called "venting" of the subarachnoid space is largely due to the vertebral veins.

Before closing the discussion on cerebro-spinal fluid pressure, I wish to refer to those cases of lumbar puncture in which the patient is postured so that his thighs press on his abdomen, and also to the restless, non-cooperative patient who makes it necessary for the assistant to place his fist firmly in the abdomen to obtain flexion of the spine, for in these cases an undue amount of blood will probably pass into the vertebral plexus with proportionate rise in cerebro-spinal fluid pressure. This may give a misleading figure.

3. Lastly, O'Connell discusses the circulation of the cerebro-spinal fluid. To the large variations of cerebro-spinal fluid pressure caused by cardiac and respiratory activity, he attributes the displacement of the cerebro-spinal fluid into the spinal theca and the region of the villi (where pressure is lowest). Also to the same cause he attributes the formation of arachnoid granulations from the microscopic villi, for O'Connell believes that the intermittent rise and fall of cerebro-spinal fluid pressure give rise to hypertrophy of these villi. Again, in post-traumatic *hernia cerebri*, the contused and weakened region of the brain is bulged by the intermittent rise and fall of pressure, and this factor is also the cause of its progressive bulging until it becomes a "hernia".

O'Connell attributes all this to the rise and fall of cerebro-spinal fluid pressure, consequent on cardiac and respiratory activity. But whereas he believes that the mechanism is the change of volume in the intracranial veins and arteries, I believe that the factor of the vertebral veins is just as great or even greater.

The work of Weed, Flexner, and Clark would seem to support the theory that the engorgement and collapse of the vertebral veins have a profound influence on cerebro-spinal fluid pressure. They have shown a striking relation between the dislocation of cerebro-spinal fluid and its pressure. According to these workers, the physiological mechanisms involved can be considered as elastic systems. These mechanisms apparently concern the elasticity not only of the anatomical coverings of the nervous system (the cerebro-spinal fluid channels), but also of the blood vascular system. Weed *et alii* state that the elasticity of the dura against outward distension seems to be very small indeed, for it is closely applied to the bony skull and constitutes a membrane of great rigidity, of much greater rigidity than the spinal dura, suspended in the epidural space, with its areolar tissue and thin-walled veins. The fact that in most animals a constant relationship exists between the fluid dislocated and the resultant pressure of the cerebro-spinal fluid indicates a considerable elasticity which permits a certain dislocation of fluid even in the intact animal. This dislocation of fluid may be due to the elasticity (collapse inward) of the spinal dura on the vertical head-down tiltings, but it also seems related fundamentally to the possible compression or dilatation of blood vessels. Weed *et alii* continue as follows. Because of the high pressure existing in the arteries of the central nervous system in comparison to that of the veins, it would seem important to emphasize the elasticity of the venous system as a factor in the establishment of the relation between the dislocation of the fluid and its pressure rather than the elasticity of the arterial channels. We have, then, in the central nervous system, a mechanism which relates the dislocation of cerebro-spinal fluid to the resultant pressure. It indicates, Weed *et alii* state, that the whole central nervous system may be looked upon as enclosed within elastic membranes, whose function is modified by the rigid character of the bony encasement. Once again we have evidence of the great part played by the intermittent engorgement and collapse of the vertebral veins in the variations of cerebro-spinal fluid pressure.

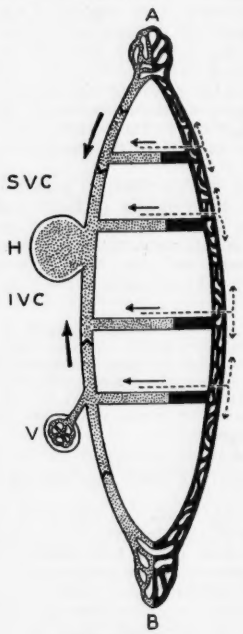


FIGURE VI.

Diagrammatic representation of the two most important venous systems in the body. The caval system is shown in stipple, the vertebral system in black. The superior polar (A) and inferior polar (B) plexuses are illustrated, and the thoraco-abdominal anastomoses are shown. The usual venous flow is indicated by black arrows, and the flow in raised cavity pressure by dotted arrows. H—heart, V—viscera, S.V.C. and I.V.C.—superior and inferior *vena cava* respectively.

Many factors of cerebro-spinal fluid physiology have been discussed. I do not discount the present theories altogether, but rather I have discussed them with a view to opening new fields of research. Also it is not my intention to discuss the merits of the papers discussed, nor do I offer a solution for all points raised. However, it does appear that a major factor, the vertebral plexus, has been much overlooked.

Not only is this blood shift in and out of the spinal canal with changes in respiration important in cerebro-spinal fluid pressure, but it proves that Harvey's statement that the blood moves in a circle is not absolutely true. This is the most significant interpretation of this phenomenon and will be discussed in greater detail in the last section.

In pathology this plexus is no less important. In fracture-dislocations of the spine, in traumatic spinal injuries in general, and especially in spinal surgery, the vertebral venous system is of immense importance. In the case of emboli, it provides a means of spread which, as Batson has stated, allows the lung capillary bed to be by-passed. In cancer spread it provides no doubt the clue to many metastases as yet unexplained. In cancer of the prostate, the constant straining on micturition and consequent raised abdominal pressure carry the metastases by the blood to the spine. In cancer and abscess of the lung, cough causes raised intrathoracic pressure, and metastases are carried to the vertebral veins; this may account for metastases in the brain. We may remember that the bronchial veins, draining the bronchi and lymph nodes, travel to the vertebral system. In connexion with neuroblastoma of the suprarenals in children, we may note that Hutchison's syndrome involves metastases in the cranium, and thus, in contradistinction to Pepper's syndrome associated with the right suprarenal, occurs classically on the left side. Poirier's description shows that the anastomoses between the renal and azygos systems occur only on the left side.

It seems, then, that just as the trunk consists mostly of two cavities, abdomen and thorax, which are undergoing constant alterations of pressure, so the pressure

relationships in the veins of the walls of those cavities likewise undergo severe alteration of pressure. When we remember that the pressure in the vertebral vein system is zero, and that the systemic vein pressure, reinforced by raised cavity pressure, is so much greater, we can realize that intermittently blood will flow into the internal vertebral plexus. We have already seen that, by the nature of the plexus of veins, it is their function to carry out the duty of equalizing the pressure of the venous system. More so, when we consider that the venous blood is so much greater in volume than the arterial blood, it appears that the circulation is capable of carrying out the task. Thus, when the venous system is taken as a whole, except for the largest vessels which contain valves to direct flow to the heart, the blood is in constant movement back and forth and in and out of the vertebral veins in accordance with the pressures in those regions. The relation with cerebrospinal fluid pressure has been dealt with, and a full realization of the problems of the drainage of the head and its various strata of veins is a much different story from that displayed in current textbooks. We can imagine that in any activity associated with increased breathing or holding of the breath, in acts of defæcation, micturition and parturition, the archaic ideas of venous circulation are untenable. The vertebral vein system is a provision of Nature to equalize pressure, to redistribute blood, and in pathological conditions of either of the two *venæ cavæ*, to act as an alternate path for the continuation of the circulation. It is important physiologically and pathologically. The portal system via the system of Retzius may have an outlet in pathological conditions of the portal vein.

It is interesting to observe that in thrombosis of the inferior *vena cava* no ascites occurs, but œdema of the back and legs is frequent. The anastomoses of the vertebral vein system have not been described in detail in this paper, as it is only an outline of some ideas on the circulation. But a study of the valves in the intercostal and lumbar veins may throw some light on the subject. Poirier mentions that Baume maintains that the intercostals at their opening into the azygos adhere intimately to the pleura, attached firmly between the ribs, so that their lumen is constantly gaping. The distribution of valves in the intercostals is remarkable. The vein in respect of valves is divided into three segments. The anterior segment contains valves which look forward, the posterior segment contains valves which look back; the middle segment has neutral valves, or is devoid of them. Thus blood is not able to circulate for the length of the vein. The blood in the middle segments is subject to two currents, one sweeping it forward to the internal mammary veins, and the other back to the azygos veins. The force drawing the blood on, Poirier and Charpy state, is inspiration and expiration. They themselves state that nothing is so variable as the ostial valves of the intercostals. They are often paired, single, atrophied, or absent. They are probably only 50% efficient, especially below, in contradistinction to those in the superior intercostals, which are better and more efficient.

FIGURE VII.
The frontispiece of Harvey's epic work on the movements of the heart and blood.

Poirier states that the valves in the lumbar veins are incompetent. Thus the veins of the walls of the thorax and abdomen contain incompetent valves and have wide open mouths. This fits in nicely with the theory I have postulated.

My last reference is to the monograph on the veins by Franklin. Some interesting facts are quoted. He states that all the blood may not necessarily be in circulation, or in rapid circulation, but part may be in reserve. In exercise the minute volume increase is eight times normal, which shows that there must be an

immense reservoir of blood. In regard to depots, he states that in exercise the following amounts are contributed by the various stores: the spleen, about 200 millilitres; the liver, probably more than 200 millilitres; and skin, about 400 to 1,200 millilitres; the lungs, a certain amount due to capillary constriction; and also a certain amount from the large uterine veins.

While we may be uncertain how much blood is contributed by the vertebral veins (if any), the point remains that as far as volume of blood is concerned it is no mean store. Batson found that 200 millilitres of dye could easily be contained in a small cadaver weighing under five stone; how much more could be contained in a living person, and one of greater stature! A glance at the atlases of Toldt and Spaltehoiz speaks for itself. Also the graphic illustrations of gross pelvic plexuses make one feel certain that between the two systems an anastomosis is certain. Franklin maintains that blood is only dammed up in expiration, and that the flow is not reversed. Yet he quotes the following experiments:

1. Naegeli and Janker show that an embolus, travelling in the inferior *vena cava*, stays at the diaphragm for a while and then ascends to the heart, but often returns again to the diaphragm before entering the heart. This seems to show that a back flow into the inferior *vena cava* occurs, despite Franklin's statement to the contrary.

2. Ribbert (1897) had similar views. He injected substances into the jugular vein and traced them back in the venous system as far as the diaphragm.

3. Haller noticed that, in animals in inspiration, air was forced back in the abdominal *vena cava*.

This illustrates again that there possibly occurs an ebb and flow with inspiration and expiration in big veins like the *venæ cavæ*. How much more so will this be so in the case of more delicate veins like the intercostals, which send the blood back into the spinal veins! It may be relevant if we mention that corsets applied to the lower half of the body of pilots gave protection against "blackout" up to a certain number of G, but after this they were a failure. The blood may have entered the vertebral veins, where it would be incompressible.

### Morphology of Veins, and the Direction of Blood Flow Within Them.

The current description of venous flow is incompatible with known facts, mainly owing to our lack of knowledge of the vertebral venous system, and owing to failure to recognize the function of this system from its architecture. Had Hilton applied his commonsense principles to the other areas of the body than the cranium, or had a disciple, keen on observation and divorcing detail from gross anatomy, followed his example, the description of the venous system would be in a better state than it is today. The current description of veins is woven around that of the arteries; this description is largely incorrect, as the function of one is active and distributing, and the other is passive and collecting. For different functions, we find different designs in anatomy. So the design of the venous system should be, and is, different from the design of the arterial system. In other words, our conception of the design of the venous system (based on the arterial system) is for the most part incorrect. Hilton was quick to realize this difference in arterial and venous architecture in the cranium, where the difference is pronounced and obvious; but, for the other regions, this false analogy was to remain for years, and has led to the mistaken conception of the venous system today. Thus we must realize that the architecture of the two systems is totally different, being determined by different physiological functions.

The arteries of the body, whose function it is to distribute blood, radiate from the heart to the periphery. The arteries resemble the spokes in a wheel, or the radii of a circle. There is some anastomosis of the arteries at the periphery, but not centrally. But this design is unsuitable for the venous system for several reasons. It is not suitable because there is no pump, and again because the venous system is a collecting system, which demands plentiful anastomoses. The veins are laid out in strata in the body. These strata resemble concentric circles around a centre or heart, and if the arteries were to be the radii of such circles, the veins and arteries would have to cross at right angles. Figure I represents the design of the two systems; but as the heart is not in the true centre of the body and is really eccentric (see later), Figure II is the more correct diagram.

The venous strata are five in number, and correspond to the strata of tissues (Figures I, II, III). (i) The veins of the central nervous system represent the deepest layer, the cerebro-spinal stratum. (ii) the epidural veins are the next deepest. These are the cranial sinuses and the internal vertebral plexus, both of which lie in the same morphological plane. The dura is composed of two layers, which split to include the cranial sinuses. At the *foramen magnum*, the inner layer descends as the spinal *dura mater*, and the outer layer continues as the pericranium, with the result that the cranial sinuses and the internal vertebral plexus are in the one morphological plane. (iii) The vessels of the axial skeleton—the diploic veins of the skull and the veins within the bony substance of the vertebræ (intravertebral veins)—form the next stratum. (iv) The veins of the "active" stratum are more superficial still. This is the plane of the soft tissues below the deep fascia, and thus is the largest or most massive in the body. It contains the body cavities and comprises muscles and viscera. Included in this are many plexuses and great vessels. The subaponeurotic plexus of the scalp, the suboccipital plexus, the pharyngeal plexus, the plexuses around the pelvic viscera, the veins of the viscera and the great venous trunks themselves are but a few. (v) The most superficial stratum is the stratum of subcutaneous veins.

If we refer back to previous statements, it is more correct to state that the "centre" of these venous strata is really the central nervous system, not the heart. The heart itself lies in the fourth stratum, and from this fourth stratum the arteries radiate. Reference to Figure II illustrates the more correct interpretation. The superficial stratum illustrates well the general principles involved. That is, it is obvious in this layer that the veins lie parallel to the surface and that the arteries are perpendicular to this same surface.

This new interpretation of the two systemic designs is not unfounded. Embryologically, the arteries commence centrally and move out to the periphery as definite channels. But on the other hand, veins occur as strata of networks or plexuses, those more superficial appearing first. In each stratum the blood flows in the network, favouring one or two channels more than the others which make up the plexus. A track is worn in the plexus, and then the vessels forming this track dilate, and the bends straighten out, with the result that definite channels or vessels appear in each plexus (Figure IV). This peculiarity of the venous system, whereby vessels appear in the plexuses, accounts no doubt for the less constant position and "make-up" of veins, or as we may prefer it, it accounts for the marked variations of veins. In the embryo are three main groups of longitudinal channels, all freely anastomosing. They are the supra-cardinal, sub-cardinal and post-cardinal veins. From these main channels and their associated plexuses the veins of the future adult arise. For example, the inferior *vena cava* chose the following course. From the caudal regions it took the post-cardinal channel, then it followed the supra-cardinal channel, and later still it entered the sub-cardinal channel. Not yet at its end, it joined the "caudal extension" by means of which it reached the heart. So the venous system must be regarded as a series of strata, each stratum being a plexus of veins extending throughout the morphological plane in which it is present. In these plexuses, channels or vessels may arise by the passage of blood favouring certain paths, with consequent variation in position and constitution of the veins in different subjects. Whether vessels appear in these plexuses depends on physiological needs in each individual stratum. It is important, therefore, to distinguish between the arterial and venous designs.

ILLUSTRATION TO THE ARTICLE BY DR. WILLIAM F. HERLIHY.

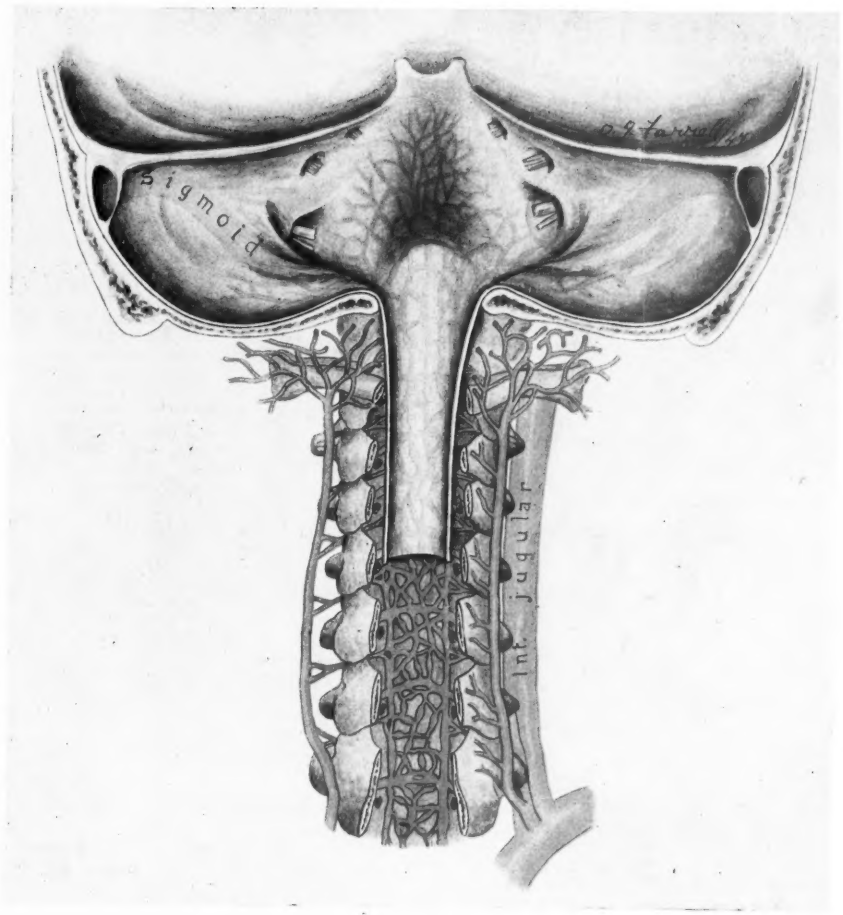


FIGURE VIII.

Illustration of the internal vertebral plexus and its anastomoses. The laminæ of the vertebræ and the posterior part of the skull have been removed, along with the brain, spinal cord and meninges, except the dura of that portion of the skull remaining and that part of the spinal dura that lies opposite the vertebral bodies. The spinal dura has been removed below the fourth cervical vertebra to show the plexus in detail. The internal vertebral plexus with its dilated vessels is shown, and also its extension onto the clivus where it anastomoses with the sinuses at the base of the skull. The two anterior longitudinal channels and the transverse annular veins, opposite the bodies and into which the basivertebral veins of the vertebral body substance drain, are depicted. When engorged, the plexus completely hides the vertebral bodies. Anastomotic vessels, passing through the intervertebral foramina to anastomose with the deep cervical veins (depicted) and vertebral veins, are shown. On the laminæ is seen the posterior external vertebral plexus (the anterior external vertebral plexus lies on the front of the bodies). The left deep cervical vein is drawn laterally for pictorial purposes. The right vertebral vein is shown only in its lower part, passing in front of the two lower transverse processes.

It must always be remembered that each stratum anastomoses with the strata above and below; in fact, it would be better to regard the divisions of strata as being arbitrary only, for each stratum merges imperceptibly into the adjacent stratum. The connecting vessels may be obvious or not. Vessels in one layer may sink down to meet the layer below—for example, the external jugular vein deepens to meet the great vessels or the root of the neck. The emissary veins of the skull are well known. But even where no definite vein is obvious connecting the strata, there is an anastomosis between them by the merging of one plexus into the other.

If, then, the venous system is plexiform in structure, how do we account for the great venous canals? There are two explanations, the first being the maintenance of the circulation, the second being the presence of body cavities.

All the great vessels, the *venæ cavæ*, jugular veins, *et cetera*, occur in the fourth or "active" stratum. For the continuance of the circulation, blood must return to the heart, and as this organ lies in the active stratum, it is only natural that this stratum would be devoted to the elaboration of a system of collecting vessels. Thus we find the big collecting trunks in this layer. This layer is called the "active" stratum, for it contains the most active organs (muscles, viscera, *et cetera*). Also because these organs are more "active" than those of other strata, it is not surprising to see that their blood supply is voluminous. Thus we find great plexuses surrounding various organs (for example, the rectum, the uterus, the thyreoid, the vagina, the prostate) coping with the huge blood supply so vital to the organ for metabolism. It is no wonder, then, that this great pool of venous blood was diverted into channels to return to the heart. It was providential then that the heart was formed in this "active" stratum, for a voluminous amount of blood is present which is easily utilized to maintain the circulation. The greater need of blood of these organs keeps open their embryonic network of vessels, and we may also note that the similar network at focal points—where several tributaries collect—is kept open. Thus we have the great prostatic, uterine and vaginal plexuses on one hand, and the great facial and pterygoid plexuses on the other. No single vessel arose here in these regions. Thus it was an easy task for Nature to get hold of this rich supply of blood for the heart. Travelling at first through the network, the blood then developed channels, and subsequently vessels in the plexus, whereby it was returned to the right atrium. I have already discussed the formation of the vessel called the inferior *vena cava*. So that the flow will be in the direction of the heart, valves developed in the vessels, and the movements of the muscles and the pressure changes in the pleural and abdominal cavities are harnessed as a motor force.

Already I have mentioned the formation of vessels leading back to the heart. But we must consider the effect of the formation of body cavities. As in Figures IV and V, the active stratum splits into two, the developing cavity compressing the plexuses in the walls of the cavity, until, when the thoraco-abdominal cavity is fully developed, the plexuses in the wall have been almost obliterated. Eventually the plexuses become obliterated, and the several channels in the plexuses become united into one main channel—the *venæ cavæ*, portal vein, *et cetera*—and are pushed to one or other side, with the result that they usually lie adjacent to the arteries. This gave rise to the present-day conception that the venous system is in design much the same as the arterial system. However, in those regions not affected by the body cavities, the more primitive plexiform arrangement persists, unassociated with arteries. Thus, in the active strata, plexuses persist in the neighbourhood of viscera and the portions not related to the body cavities. But in the region of the body cavities large vessels have developed, which connect the poles of the organism so that the circulation may be maintained.

It has now been made clear that it is the active stratum in great part that supplies the venous blood to the heart. It must be realized that the other strata are concerned only indirectly with this function. It must be pointed out here, that with the great development of the brain in man, the cranial sinuses are large, and at the base of the skull the blood within them is directed into the active stratum (internal jugular vein). Again, if cardiac return and output are equal, it becomes obvious that as the venous system contains much more blood than the arterial system, Harvey's idea that all the blood was in continual circulation is incorrect. That is, a great portion of venous blood at any given moment is not concerned with circulation.

Returning to our five strata, we can now state that four are mainly plexiform and that only one has a great development of vessels. Of these five, we can also state that two are more important than the others—the caval system, which consists of trunks to return blood to the heart, and the vertebral venous plexus, which retains its plexiform structure and forms a giant anastomosis connecting the poles and sides of the caval system (Figure VI).

Its extensive anastomosis and plexiform arrangement make the vertebral plexus so important in the body, for it is by means of this plexus that venous pressure is regulated, *et cetera*. The blood in the vertebral plexus is not primarily concerned with circulation.

With regard to the pulmonary, coronary and portal systems, all that will be said is that they have been developed in the active stratum for various collateral purposes, and are to be regarded as subdivisions of the caval system, or better still, as adjuncts to it.

So we must regard the venous system as being composed of five strata, of which the main two are the caval and vertebral systems. The former is associated mainly with the maintenance of venous return and the circulation, the latter functions by nature of its wide anastomoses.

To return to the conception of Harvey: he believed that the blood flowed into the arteries and back to the heart through the veins in a continuous circle. We now see that this is not correct.

I believe that the blood is distributed by the arteries and capillaries to the body in the manner usually described. But the collecting system consists of multiple strata of veins, in which there is a tendency for the blood to flow centrally to the heart. However, as there is more blood in the venous system than in the arterial system, all the venous blood has not to return to the heart immediately (as Harvey inferred). The circulation will be maintained if venous blood, equal in amount to the cardiac output, is returned to the heart at similar intervals. The venous blood destined for maintenance of circulation mainly exists in the active stratum, and is collected for this purpose by the great veins. The remainder of the venous blood lies in the other strata, moving centrally; but with regard to these other strata I wish to draw special attention to the pool of venous blood in the vertebral veins. The functions of this plexus—equilibrization of pressure, reception of backflow and redistribution with respiration, *et cetera*—have been mentioned. In and out of this plexus blood runs, not unlike the earliest conceptions of an "ebb and flow".

The major divisions therefore of the venous system are (*a*) the caval system, for maintenance of circulation, and (*b*) the vertebral system, which in virtue of its size, position and countless anastomoses, possesses functions as described above. Eventually all the venous blood returns to the heart, but not in a continuous circle, as Harvey would have. The realization of this is of the utmost importance in anatomy, physiology, pathology and surgery.

## SUMMARY.

Our present conception of the venous system is incorrect. It is really composed of five strata, of which the caval system (fourth stratum) and vertebral system (second stratum) are the most important. The physiology and pathological implications of the vertebral system are described. I again wish to state that this work is only an hypothesis, but it is put forward as such for critical study and examination.

## ACKNOWLEDGEMENTS.

I am indebted to Professor A. N. Burkitt for his guidance in the preparation of the paper, and to Professor

J. Shellshear for his invaluable suggestions, and I wish to accord my thanks to Miss Hunter, librarian of the Department of Anatomy, University of Sydney, and to my colleague, Dr. Wyke, for his invaluable help. All diagrams were drawn by Mr. D. Farrell, and I here record my appreciation of them.

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## A CLINICO-PATHOLOGICAL STUDY OF TWO CASES OF IDIOPATHIC CARDIAC HYPERTROPHY WITH CONGESTIVE FAILURE.

By MALCOLM FOWLER,
*From the Department of Pathology of the University of Adelaide.*

THE disease known as idiopathic cardiac hypertrophy was first described by Josserand and Gallavardin[1] in 1901. Within recent years numerous examples of the condition have been described in adults, including a series of ten cases by Levy and von Glahn,[2] five cases by Reisinger and Blumenthal,[3] and eleven cases by Kaplan, Clarke and de la Chapelle.[4] In addition, Kugel[5] and Kugel and Stoloff[6] and others[7][8] have reported a similar but not necessarily identical disease in infants.

During the past year two young men have died from the condition at the Royal Adelaide Hospital. The clinical and pathological findings are here recorded.

### Reports of Cases.

CASE I.—J.B., aged twenty years, single, an engineer's apprentice, was admitted to the Royal Adelaide Hospital on August 29, 1946, under the care of Dr. A. R. Southwood. For two months he had suffered from dizzy turns, and for one month from pain in the left side of the chest. During the week before his admission to hospital this pain had been continuous. Breathlessness on exertion had been present for an indefinite period. His sleep had been undisturbed. He had coughed up a quarter of a pint of blood the night before entering hospital.

On examination, the patient was seen to be a pale youth in some slight respiratory distress, with a pulse rate of 112 per minute and a normal temperature. The blood pressure was 115 millimetres of mercury (systolic) and 90 millimetres (diastolic). The maximal impulse of the heart was in the sixth intercostal space four and a half inches from the mid-sternal line. The urine was normal according to the usual ward tests. No special investigations were carried out.

The patient died after a sudden increase in dyspnœa three days subsequent to his admission to hospital.

#### *Post-Mortem Examination.*

Autopsy (Post-Mortem Examination Number 212/46) was carried out fifteen hours after death by Professor J. B. Cleland.

*Macroscopic Examination.*—The body was that of a fairly thin young man of normal appearance. The heart weighed 830 grammes and was greatly hypertrophied and somewhat dilated in all chambers. Large ante-mortem thrombi had formed on the anterior wall and apex of the left ventricle. At the apex the myocardium was considerably thinned. Neither valvular defects nor coronary disease were present. The endocardium was milky in colour in all chambers. In

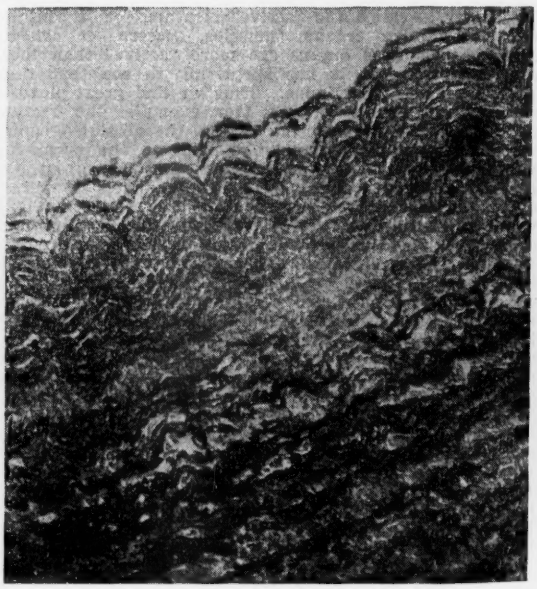

FIGURE I.
Case I; fibro-elastic proliferation in endocardium (hæmatoxylin and eosin, × 540).

the lungs were two small infarcts, one measuring two and a half inches in diameter, the other one and a half inches. The lungs, spleen, kidney and liver were congested, the last-mentioned having a typical nutmeg appearance. The thyreoid, pituitary and suprarenal glands were normal. The thymus weighed 23 grammes.

*Microscopic Examination.*—The endocardium throughout was much thickened. Immediately subjacent to it there was a great increase in fibro-elastic tissue, extending into bundles of degenerating muscle (Figure I). In the myocardium were found patchy areas of necrosis which appeared as pale granular material mixed with disintegrating muscle fibres. Various stages of fibrosis were present throughout the muscle. Sometimes the fibrous tissue penetrated between individual necrotic muscle fibres. There was no evidence of inflammatory reaction. Other muscle fibres were less affected, but their cytoplasm contained deeply staining granular material. The majority of the muscle fibres had large hyperchromatic nuclei, distorted into many bizarre shapes. Often the nuclei were lobed or spider-like, and invariably they had one concave edge. Many of the muscle cells were separated from their sheaths by a clear space, the appearance suggesting œdema. The muscle fibres measured from 17$\mu$ to 33$\mu$. For the most part the blood

vessels were normal, but occasionally there was slight perivascular infiltration with lymphocytes (Figure II). One small artery contained a partially canalized embolus. The other organs showed evidence of venous congestion. No changes were found in the kidneys, nor were the small arteries of the body thickened.

CASE II.—N.P., a schoolboy, aged fourteen years, was admitted to the Royal Adelaide Hospital on February 28, 1945, under the care of Dr. Guy Lendon. Twelve days before he had had a dizzy attack and had gone to bed; his doctor found that he had a temperature of 101° F. and a discrete macular rash on his back. A sore throat had been present for two weeks, but he had no joint pains.

On examination of the patient, the heart was not enlarged, but was beating with a triple rhythm and a systolic murmur heard best in the mitral area. The blood pressure was 130 millimetres of mercury (systolic) and 100 millimetres (diastolic). The blood sedimentation rate was 60 millimetres (first hour) and 105 millimetres (second hour). The electrocardiogram revealed no abnormality. The urine was normal. The red blood cells numbered 3,950,000 and the leucocytes 10,200 per cubic millimetre, the differential count in the white series giving normal results. The patient's temperature on his admission to hospital was 101° F.; under salicylate treatment it fell steadily. He was discharged two months afterwards.

FIGURE II.

Case I; infiltration of lymphocytes about an artery, with some surrounding degeneration of muscle fibres (hæmatoxylin and eosin, × 250).

Five months later the boy was readmitted to hospital with a history of shortness of breath on exertion, shivering attacks and pains in both elbows and knees, all of two days' duration. The dyspnœa was worse at night and was accompanied by sweating. On examination of the patient, his blood pressure was 110 millimetres of mercury (systolic) and 60 millimetres (diastolic). The apex beat of the heart was in the mid-clavicular line. A harsh systolic murmur and a mid-diastolic murmur, together with triple rhythm, were heard at the apex, while a pre-systolic bruit was recognized by some clinicians. The average pulse rate was 115 per minute. The temperature of 101° F. was present for two periods of a week, a week of normal temperature being interposed between them. An attempt at culture of micro-organisms from the blood gave negative results. The blood sedimentation rate was raised throughout the illness. The electrocardiogram showed a prolonged *P-R* interval of 0·27 second.

The boy became short of breath and restless; his liver descended to the umbilicus, and fluid collected in his legs and peritoneal cavity. Death occurred while he was in this condition six weeks after his admission to hospital.

*Post-Mortem Examination.*

Autopsy (Post-Mortem Examination Number 235/45) was performed twelve hours after the patient's death by Professor J. B. Cleland.

*Macroscopic Examination.*—The body was that of a well-developed boy of normal appearance. The heart weighed 570 grammes. It was much dilated and hypertrophied in all chambers, without accompanying valvular or coronary disease. The endocardium of the left auricle and ventricle was opaque, evidently owing to fibrosis. This opacity was more pronounced in the auricle, diminishing towards the apex, which was normal. The endocardium of the right ventricle and auricle was also normal. In the lungs and liver chronic congestive changes were apparent. Forty ounces of fluid had collected in the peritoneal cavity, with much œdema of the trunk and limbs. The cortex of the kidneys was swollen and pale buff in colour. The suprarenal, thyreoid and pituitary glands were normal.

*Microscopic Examination.*—The only pathological changes in the lungs and liver were due to chronic venous congestion. The kidneys presented considerable cloudy swelling with no evidence of glomerular damage. No fatty changes in the tubules were found by staining or by crossed Nicoll's prisms. The endocardium of the right side of the heart was normal, whereas the lining of the left side was much thicker than usual, especially in the auricle, where it measured two millimetres in depth. This auricular layer (Figure III) was composed of collagenous and elastic fibres, with pieces of degenerated muscle and isolated muscle nuclei amongst them. Directly subjacent to this the muscle fibres showed various stages of vacuolation. Perivascular collections of lymphocytes were often present, but there was no general infiltration with these cells. Some of the small vessels near the degenerating area had swollen intimal linings and endothelial proliferation, so that the lumen was much narrowed but never occluded (Figure IV). Deeper in the muscle of the left auricle an occasional larger arteriole was often affected. The muscle fibres were smaller than would be expected, the majority measuring less than 17$\mu$, a few nearly 30$\mu$. Some had distorted nuclei, but this condition was less prominent than in the first case. The changes described above were most pronounced in the left auricle; they gradually decreased in severity down to the apex, which was healthy (Figure V). This suggests that the process had started in the auricle. Unfortunately the pulmonary veins were not available for examination.

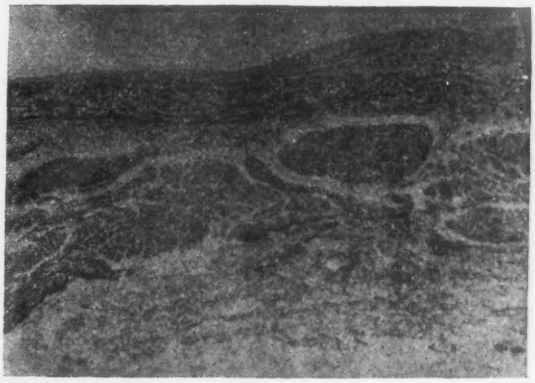

FIGURE III.

Case II; thickened endocardium of the left auricle (hæmatoxylin and eosin, × 30).

## Discussion.

Both patients were young males, one aged twenty years, the other aged fourteen. If cases occurring among infants are excluded, the ages of the patients in 28 cases recorded in the literature ranged from eleven to sixty-seven years. There was an approximately equal distribution in the age groups, except between forty and fifty years, where the incidence was doubled.

One striking feature of the series of 28 cases referred to above was that only three of the patients were females. This may indicate a sex-linked factor, an inherited inferiority of the cardiac muscle.

The first of my patients had no history of infection. On the other hand, the second patient first fell ill with an undiagnosed infection characterized by a sore throat, a raised temperature and a discrete macular rash on his back. The previous history of other recorded cases contained no significant features, nor was there any factor precipitating the often sudden onset of cardiac failure. Levy and Rousselot[9] described the cases of two men who complained of past attacks of tachycardia; Whittle's patient[10] had tracheitis; while in some American cases there was a history of syphilis or gonorrhœa.

The clinical findings in my second case were especially interesting in view of their close resemblance to some of the features of acute rheumatic fever. The occurrence of cardiac murmurs, the raised blood sedimentation rate, the anæmia, and finally the pyrexia which responded to salicylate treatment, were all suggestive. The symptomatology on the patient's readmission to hospital seemed even more conclusive—joint pains, loud systolic and diastolic murmurs, and an increase in the *P-R* interval. The fact that

FIGURE IV.

Case II: a small vessel in the subendocardial region showing endothelial swelling (hæmatoxylin and eosin, × 250).

the malady may simulate acute rheumatic fever has not previously been recorded.

In the majority of cases the first symptom was breathlessness increased by exertion; but two of Levy and Rousselot's patients[9] first complained of palpitation. Chest pain as the earliest symptom was described in the case records of the Massachusetts General Hospital[11] and by Doane and Skversky.[12]

In some of the reported cases there was a slight elevation of temperature associated with infarction of the lungs, although (as in Case II) the reason for this pyrexia in a few was obscure.

Frequently there was evidence of embolism, including sudden blindness in one eye,[9] and hemiplegia;[2][12] pulmonary embolism was common.

Both of the patients under discussion had regular pulse rates of over 110 per minute; but Reisinger and Blumenthal[3] and Levy and von Glahn[2] each observed three patients with auricular fibrillation. Cases of complete heart block were recorded in the case records of the Massachusetts General Hospital[13] and in the paper by Levy and von Glahn,[2] while partial block was reported by Levy and Rousselot.[9]

Whether the disease is familial remains undetermined. In this connexion it may be mentioned that one of the patients referred to by Smith and Furth[14] had a sister who died with a vague diagnosis of myocarditis of unknown origin. The case records of the Massachusetts General Hospital[13] contain similar, but equally inadequate, references. Sprague, Bland and White[8] described the occurrence of congenital idiopathic cardiac hypertrophy in an infant, heart disease of some kind being present in most of the family.

The duration of life from the first symptom was short in the two cases described, the first patient living two months, the second eight months; but according to other writers the period is variable. Whittle[10] and Reifenstein and Chidsey[15] each recorded a case of a young boy who dropped dead without previous symptoms. In other instances the patients had been ill for several years. One patient, reported in the case records of the Massachusetts General Hospital,[16] had had an attack of cardiac insufficiency when five years old and dropped dead six years later. However, with the onset of breathlessness, in most of the cases the condition followed a swift and unremitting course.

The part played by antecedent high blood pressure (for example, Case I) is often debatable; but too often there

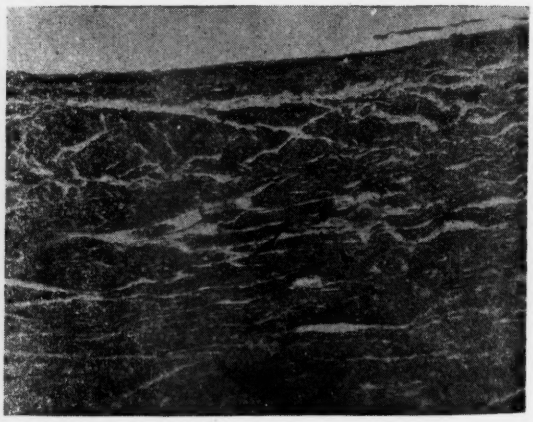

FIGURE V.

Case II; slight thickening of the endocardium of the left ventricle (hæmatoxylin and eosin, × 30). Compare Figure III.

is a tendency to blame earlier hypertension even when evidence of it is lacking. Kaplan, Clarke and de la Chapelle[4] compared the kidney arterioles and preglomerular capillaries in 43 cases of idiopathic cardiac enlargement with the same structures from a normal group of subjects of the same age and found no evidence of antecedent hypertension.

The first patient of the present series was under observation for only three days, and throughout this period his blood pressure was normal. Thus the possibility remains that he may have had previous hypertension which subsided with the onset of cardiac failure. But, if such hypertension, either by degree or duration, could cause an increase in heart weight to nearly three times the normal figure for a man of twenty years, one would expect other changes typical of hypertension, such as thickening of small arteries, especially those in the kidney.

The pathological changes seen in the heart vary considerably, but this variation seems to have no relation to the clinical course. In their case Reifenstein and Chidsey[15] found no lesions except hypertrophy. On the other hand, Mahon,[17] in an excellent description of cardiac hypertrophy in two children, discovered widespread endocardial fibrosis, with granular degeneration of muscle fibres and swelling and proliferation of the endothelial cells of small arteries in the heart. Diffuse scarring of the

myocardium was the main feature in some cases. In one of Levy and Rousselot's cases[9] hydropic changes of the muscle fibres were present, together with an unusual form of nuclear degeneration.

Earlier investigators found that the subendocardial muscle often suffered most. In the second of the present cases damage was restricted to this layer. Another common finding was the occurrence of plaques of fibro-elastic proliferation of the subendocardium often associated with ante-mortem thrombi, as in Case I in this paper.

In Case I, on microscopic examination, an embolus was discovered in a small artery in the heart, but it was too small to account for the widespread changes. However, there may have been multiple emboli of the same kind which were not found; but this possibility is remote and does not explain the formation of the mural thrombi in the first place. Other embolic phenomena are described, such as infarcts in the lungs, kidneys and brain, one death being due to coronary embolism.[26]

Changes in the other organs due to chronic venous congestion were detected. No other lesions were unearthed which could explain the cardiac hypertrophy.

An interesting point is whether the muscle fibres are really hypertrophied. In his cases in children Mahon[17] showed that this was not so. Similarly, in the present cases, the fibres had not hypertrophied, and in one case they were actually smaller than normal when measured by a micrometer. Karsner, Saphir and Todd[18] have shown that enlargement of the heart occurs by hypertrophy of muscle fibres, but these cases suggest another factor.

Various hypotheses have been put forward to explain the pathology of this disease. The remarkable feature about it is the complete absence of any cellular reaction to the degenerative process which is so conspicuous in such conditions as rheumatic fever and Fiedler's myocarditis.[19][20] Even if the disease was a rheumatic infection without valvular involvement, one would need to see the characteristic Aschoff lesion or the more diffuse cellular infiltration for such a diagnosis to be made. Many blame an enlarged thymus, as in Whittle's case,[10] although the significance of such enlargement has not been determined.

Some writers, including Smith and Furth,[14] have suggested that these cases may be due to a deficiency of vitamin *B* complex, although their patients made no convincing improvement under treatment. The two patients reported here presented none of the accepted features of a deficiency, such as polyneuritis, epithelial ulceration or dermatitis. Follis and others[21] have produced cardiac lesions by feeding rats on diets deficient in potassium. The early lesions induced were necrotic areas in muscle fibres with a leucocytic reaction. These degenerated areas were later converted into scar tissue. The application of this experimental work to human subjects has not been defined.

The question of von Gierke's glycogen storage disease was considered, but the usual appearance of the heart showing a cytoplasmic network of vacuolated muscle fibres, as described by Antopol, Heilbrunn and Tuchmann,[22] was not present. Most cases of this disease occur in young children, and other organs are usually affected as well as the heart.

Flynn and Mann[23] have shown recently that endocardial degeneration often occurs in patients dying of congestive cardiac failure. They demonstrated concomitant thrombosis of the Thebesian veins, which they considered was secondary to mural thrombosis. Notwithstanding this, my second patient had neither mural nor venous thromboses, although in the Thebesian veins degenerative changes were observed. I suggest that thrombosis of the Thebesian veins occurs first, with consequent subendocardial degeneration, and then mural thrombosis.

The two cases presented may be variants of the same disease entity, the first, despite the shorter history, being the more advanced form. This condition seems to be a progressive fibrosis of the heart from within outwards; in the second case this started in the left auricle.

The theory could be advanced that the noxious agent, entering the circulation through the lungs, attacked the auricle (via the Thebesian veins) and, less virulently, the ventricle, where these veins are less plentiful. The progressive character of the disease could be explained by the assumption that the fibrosis following thrombosis in the Thebesian veins caused interference with the coronary capillary anastomosis, and so increased the peripheral resistance within the coronary circulation. This would explain the endarteritis in the small vessels, a change which would lead to further fibrosis.

## Summary.

Two cases of idiopathic hypertrophy of the heart with congestive failure are described. The patients were both young men, aged twenty and fourteen years, and the hearts weighed 830 and 570 grammes respectively. The younger subject presented a clinical picture simulating acute rheumatic fever.

Pathological examination disclosed endocardial thickening and necrosis of muscle fibres; in one case lesions were confined to a subendocardial layer only.

Brief reference is made to other reported cases, especially to the predominance of the disease in males.

The bearing on the ætiology of the condition of antecedent hypertension, of vitamin *B* complex and potassium deficiencies, of glycogen storage disease and of thrombosis of the Thebesian veins, is discussed.

## Acknowledgements.

I am indebted to Dr. A. R. Southwood and Dr. Guy Lendon for the clinical notes, and to Professor J. B. Cleland for the gift of his pathological material.

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### Addendum.

Since the compilation of this manuscript, R. F. Norris and H. H. Pote have published a paper entitled "Hypertrophy of the Heart of Unknown Etiology in Young Adults: Report of Four Cases with Autopsy" in the *American Heart Journal*, Volume XXXII, 1946, at page 599. All four patients were young men, and the pathological findings were similar to those described above.

## Reviews.

### TEXTBOOK OF GYNÆCOLOGY.

THE second edition of a "Textbook of Gynæcology", by J. H. Peel, appears only three years after the first edition.[1] The general design of this excellent book remains the same, but much of the text has been revised and rewritten and some new illustrations have been added. The section of endocrinology has been brought up to date and other recent advances in treatment have been included. The sections on ætiology, physiology and the endocrine disorders have been thoroughly revised, and incorporate the latest ideas and the modern teaching in these subjects. The endocrine section conforms with the opinions and teaching held by the British school, and the danger of the haphazard and indiscriminate use of hormones is pointed out. There is also an excellent appendix of the endocrine products, and this should be of invaluable help to many students and practitioners who are confused with the nomenclature and action of the various hormones marketed by the pharmaceutical houses in this country. Other valuable sections on symptoms and their treatment in gynæcology are well written, and include a detailed description of medical, physiotherapy, radium and X-ray treatment, including the modern advances in these subjects. Actually medical gynæcology receives more attention than is usually given in a textbook of this size, and it is gratifying to see the importance of medical treatment brought into such prominence.

The only adverse criticism of this book has to do with the section on anatomy and embryology. From the student's point of view this section will require supplementing, and it is a pity that a book which carries such detail on other aspects of gynæcology should not find more space for this important section. There are a few irritating printer's errors in the early sections.

This book can be recommended to students and practitioners as a concise, orthodox and well-written textbook in up-to-date gynæcological teaching.

### PULMONARY TUBERCULOSIS.

THE second edition of "Pulmonary Tuberculosis", by R. Y. Keers and B. G. Rigden, a "concise" handbook, has appeared within two years of the earlier edition and manitains its high standard.[2] The authors point out that no major developments have occurred in the field of medicine in this time. Perhaps not; but progress at least from the scientific side marches on and the results of measures of treatment, whether designed to assist Nature's own healing process, such as routine sanatorium care, supplemented with mechanical means of treatment and chemotherapy, must surely be assessed over decades. The authors have done well to include aspects of Rich's work on pathogenesis and to refer to some of the problems of rehabilitation and plans designed to help the patient after he leaves a sanatorium. This, of course, is a subject with wide ramifications in society and might well occupy a complete treatise.

The authors state that "it can now be fairly claimed that pneumoperitoneum has found a place for itself among the methods of collapse therapy at our disposal". Pneumoperitoneum is still under trial, and many are not convinced of its value as yet. Further time will probably be required to assess its merits.

As in the previous edition, the book is well illustrated with excellent skiagrams, and with each is drawn a diagram to show the lesions present. These are undoubtedly of very great help to the student or doctor not familiar with the interpretation of films. They are well worth the time and trouble which were necessary in their preparation.

[1] "Textbook of Gynæcology", by J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G.; Second Edition; 1946. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 484, with many illustrations. Price: 21s.

[2] "Pulmonary Tuberculosis", by R. Y. Keers, M.D., M.R.C.P., (Edinburgh), F.R.F.P.S. (Glasgow), and B. G. Rigden, M.R.C.S. (England), L.R.C.P. (London), with a foreword by F. H. Young, O.B.E., M.D. (Cambridge), F.R.C.P. (London), D.P.H.; Second Edition; 1946. Edinburgh: E. and S. Livingstone, Limited. 7¼" × 5", pp. 305, with many illustrations. Price: 17s. 6d.

## Notes on Books, Current Journals and New Appliances.

### STORIES OF A STATESMAN.

IN "Crusts and Crusades" Mr. William Morris Hughes tells some stories out of his long and varied life.[1] They deal with his own personal experiences, particularly in the early days of his career when he came to Australia a stranger and had to face deprivations and hardships of all kinds. He throws some light on the early days of the Labour Movement. He was full of enthusiasm and was also gifted with a great sense of humour. He tells no stories about his parliamentary colleagues because this would be telling tales out of school and a breach of the code adopted by him in public life. His story of what happened at the sessions of the Reparations Commission after the war of 1914-1918 is the last in the book; it ends with the grievances of Van den Henvel, the Belgian representative. The tale must be read as Mr. Hughes has written it. Australians of every political persuasion should read this book.

### TWICE A PRISONER OF WAR.

IT is not often that it falls to the lot of a soldier to be taken prisoner twice in one war, but this was the unfortunate experience of Lieutenant Walter Summons, of the 2/2 Pioneer Battalion, Australian Imperial Force, and is told by him in "Twice Their Prisoner".[2] He began his first term of captivity in 1941, when he was taken prisoner by the French in the campaign in Syria. He and his fellow prisoners were taken by air from Aleppo to Salonika in Greece, and from there began a train journey through Yugoslavia, Austria, Germany and France to Toulon. From Toulon the party was repatriated to Beirut by sea about three months after their capture.

Not many months afterwards Summons and his unit were on board the *Orcades* on their way to Sumatra and then to Java, and here on the surrender of the British and Dutch forces began his second captivity. He was one of the many men who were sent to work on the Burma-Thailand railway.

Mr. Summons enlisted when little more than a schoolboy and while still under age, and his style of writing is that of a youth who sets down his experiences in a plain and completely objective manner; he is not an introspective type, and his book is mainly a record of his life as prisoner; his thoughts, as far as he tells us, were mostly worries over his family at home and of his dislike of captivity. But the thing that stands out in his pages is the courage of spirit displayed by the men who slaved under these appalling conditions. And he pays a wonderful tribute to those medical officers who never slackened in their struggle to save the lives of the men under their care.

The book is well illustrated with photographs and drawings.

[1] "Crusts and Crusades: Tales of Bygone Days", by the Right Honourable W. M. Hughes, C.H., K.C., M.P.; 1947. Sydney, London: Angus and Robertson, Limited. 8½" × 5½", pp. 246. Price: 8s. 6d.

[2] "Twice Their Prisoner", by W. I. Summons; 1945. Melbourne; Oxford University Press. 8½" × 5½", pp. 198, with illustrations. Price: 12s. 6d.

The Medical Journal of Australia

SATURDAY, MAY 31, 1947.

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## THE NEXT CONGRESS.

No matter what kind of change comes over the face of medicine in the post-war stage of social evolution, there is one aspect that will never change, one requirement that will always have to be met. This is the free interchange of ideas by formal discussion and by informed talks between small groups of persons. As knowledge grows and the scientific basis of medicine becomes more complex, the interchange of ideas becomes more and more necessary. This is something more than the talking of "shop", to which medical folk are said to be addicted more than most people. The talking of "shop" is sometimes a desultory business and may be nothing more than a recital of somebody's interesting cases. Even such shop may provide food for thought, but the ideas referred to are concerned with fundamentals rather than with final details, with the "how" and the "why" rather than with what happens and what to do when it does happen. Hospital staff meetings and British Medical Association Branch meetings are an expression of the interchange of ideas of this kind and in pre-war days the Australasian Medical Congresses were the logical and natural extension of these meetings. Here was the stimulus of a large gathering of men and women from different centres of learning and practice whose point of view varied with their environment. It is not to be wondered at that success has attended each of the five sessions held since 1923, when the British Medical Association assumed control of the congresses. After the interruption caused by the recent war congress will meet once more, and the announcement that the sixth session is to be held at Perth in August, 1948, has been acclaimed in every State of the Commonwealth.

This is the third attempt of the Western Australian Branch of the British Medical Association to hold a congress in Perth. The first attempt had to be abandoned in the years of the financial depression; the second was interrupted by the outbreak of war in 1939. In spite of these misfortunes the keenness and enthusiasm of the members of the Western Australian Branch have not waned; if anything, they are greater than ever before. No time has been lost since the Federal Council at its last meeting accepted the invitation of the Western Australian Branch and appointed Dr. D. M. McWhae as president-elect. The Federal Council meeting was held at Melbourne in the first week of March of this year, and the first meeting of the executive committee of congress took place three weeks later. Dr. D. M. McWhae, the President, has with him on the executive Dr. H. M. Trethowan, honorary secretary; Dr. H. MacMillan, honorary treasurer; Dr. G. B. G. Maitland and Dr. L. G. Male, assistant honorary secretaries; Dr. McKellar Hall, president of the Western Australian Branch; Dr. L. E. Le Souef, past-president of the Branch; and Dr. F. W. Carter and Dr. N. M. Cuthbert, representatives of the Branch on the Federal Council. The General Secretary of the Federal Council and the Editor of THE MEDICAL JOURNAL OF AUSTRALIA are members of the executive *ex officio*. The executive committee has secured the services of a lay secretary, and the office of the congress is at Shell House, St. George's Terrace, Perth. The date of the session has been fixed at Sunday, August 15, to Saturday, August 21, 1948, and the Chancellor and Council of the University of Western Australia have generously made the university buildings available as a meeting place. The University of Western Australia, with its beautiful Winthrop Hall, is ideally placed for the holding of a congress. The architecture is unique among Australian universities and the whole place has an atmosphere which must have an effect on those privileged to study within its walls. The medical visitor to the University of Western Australia will probably wonder how William Osler would have written about it; that he would have been inspired to utter unforgettable words is certain. Osler said that a university had two functions—to teach and to think. Of the university on the banks of the Swan River it may be said that both functions should be easy of fulfilment. Being held in such surroundings, the congress should from the intellectual, scientific and social points of view take its place among the most successful that have been held in this part of the world.

The work of the congress will be carried on in sections. The executive committee has decided against the adoption of any central *motif* for the gathering. It has determined, however, that the plenary session shall be devoted to the problems of pulmonary tuberculosis. This is a subject which comes within the range of every branch of medical practice. It is one in which governments, the medical profession and the public are or ought to be vitally concerned. As readers of this journal are well aware, something is being done, but not enough. The Commonwealth Government has recently appointed a director of tuberculosis, and a move is being made to set up an Australian council for the prevention of tuberculosis. A full-dress discussion at a plenary session of congress may be calculated to arouse interest where it has not yet been awakened, and to give a fillip to those who may have become weary in their well-doing. Perhaps it should be stated—for in the long interval that has elapsed since a congress was held in Australia many may have forgotten—that medical politics find no place whatever in any of the activities of congress. The sections will deal with the following fourteen groups of subjects: (i) medicine;

(ii) surgery; (iii) naval, military and air force medicine and surgery; (iv) obstetrics and gynæcology; (v) pædiatrics; (vi) ophthalmology; (vii) oto-rhino-laryngology; (viii) public health, tuberculosis and tropical medicine; (ix) anæsthesia; (x) pathology, bacteriology and biochemistry; (xi) orthopædics and physical medicine; (xii) radiology; (xiii) dermatology and industrial medicine; (xiv) neurology and psychiatry. Of these sections, numbers (iii), (ix), (xi), (xii) and (xiii) will be regarded as minor sections and will not meet on every day of congress. Another decision that has been made is that there will be no separate volume of transactions. In the past the volume of transactions has been a difficulty for the reason that the transactions are issued to members of congress only. The result was that original work presented at congress did not reach a wide public. At the fifth session (Adelaide) in 1937 the separate volume of transactions was dropped. The "congress numbers" of THE MEDICAL JOURNAL OF AUSTRALIA contained a précis of every paper presented with the discussion. The papers were later published in full (with one or two exceptions) in this journal. In this way authors were assured of a wide circulation. The practice adopted at Adelaide will be continued at Perth.

Many other features of the arrangements remain to be mentioned. These include facilities for travel, accommodation, arrangements for the entertainment of visitors, and so on. Reference also should be made to the trade exhibition and the hobbies exhibition (which already promises to be of a high order). All these matters must be left for another occasion, when plans are more advanced. The present discussion is in the nature of a preliminary announcement. From time to time "Congress Notes" will be published in this journal, and readers are advised to keep themselves informed of what is being planned for their benefit. The chief thing to be done at this stage is to earmark August, 1948, as the time for a journey westward to learn at first hand what those in other places are thinking and doing, to stimulate others and to be stimulated. In this process old friendships may be renewed and new ones made.

# Current Comment.

## ACTIVITY IN THE TREATMENT OF FRACTURES.

THE relative merits of rest and activity in the treatment of fractures are matters of more than academic or even professional interest. They are liable to come into prominence in the medico-legal field and to demand justification for their use. Medical opinions still differ widely and a clear expression of opinion from an orthopædic surgeon of standing is welcome; though made *ex cathedra*, such an opinion is not necessarily to be accepted without question. George Perkins, in his presidential address to the British Orthopædic Association on October 19, 1946,[1] shows himself as an unashamed supporter of activity. In his discussion, which is confined to fractures not involving joints, he seeks to analyse the purpose and the effect of applying splints and to upset practices born of habit and tradition rather than of careful thought. Splintage may be considered as serving two purposes, namely, the maintenance of alignment and the immobilization of bone ends. Good alignment is naturally important, but is by no means always the most important factor in a sound functional result. While he realizes fully that patients tend to judge the result as seen on the radiograph, Perkins is of the opinion that the surgeon who obtains perfect function has little to fear in a court of law. The important point is that alignment may be maintained by a splint which does not immobilize the bone ends. To immobilize the bone ends, what Perkins calls "PURE" splintage is needed, that is, splintage which is protracted, uninterrupted, rigid and extensive. Protracted splintage tends to produce a permanent loss of joint range. There is no special objection to uninterrupted and rigid splintage. Extensive splintage is defined as that which is extended "until the splint can be angulated above and below the fracture". Without this, immobility of the bone ends is not assured. Extensive splintage is, however, in Perkins's view, the most harmful feature of "PURE" splintage, as it prevents synergic muscle action. This synergic action, he belives, preserves the power of muscle lengthening and so prevents joint stiffness. "PURE" splintage is thus undesirable in any circumstances. Perkins summarizes his arguments thus: (i) External splintage is either (*a*) alignment splintage which need not be "PURE", or (*b*) "PURE" splintage which immobilizes the bone ends. (ii) When there are muscles attached to both fragments, the muscles form a natural splint that keeps the bone ends immobile, and alignment splintage only is needed. (iii) When there are no muscles attached to one fragment, "PURE" splintage is essential to keep the bone ends immobile; typical examples are fracture of the neck of the femur, a fracture of the internal malleolus, and a fracture of the carpal scaphoid. (iv) "PURE" splintage induces so much joint stiffness that it ought not to be used unless absolutely necessary. (v) On occasions when "PURE" splintage is indicated, it is often better, because of its disabling effects, to substitute rigid internal splintage. While many will hesitate to follow Perkins's enthusiastic trend towards minimal splintage, all might well accept his suggestion that, when they are about to impose a splint on a patient, they should pause and ask themselves why they propose to use this splint, what good it will do and also what harm. In this matter it is always necessary to remember that the man behind the method is at least as important as the method itself.

[1] *The Lancet*, January 11, 1947.

## THE MECHANISM OF HEALING IN BACTERIAL ENDOCARDITIS.

ONE of the most hopeful things in medicine is to find that some new method promises even some degree of control of a disease previously considered hopeless. Subacute bacterial endocarditis has not been other than a depressing study with regard to its treatment prior to the introduction of the recent methods of chemotherapy. Penicillin in particular has raised our hopes, and some of the results have been very encouraging. This in turn has stimulated interest in the basic processes of healing, which used to be given greater prominence some years ago, but now has rather receded in favour of such questions as bacterial resistance or sensitivity. Robert A. Moore has contributed a thoughtful article written from the point of view of the pathologist.[1] His interest was aroused by an autopsy study on a young woman who died from a pelvic septicæmia, and who had vegetations of the mitral valve which were not characteristic of acute bacterial endocarditis. Through the Committee on Medical Research material was obtained from twelve hospitals and schools, derived from autopsies on patients who died during or after treatment by penicillin, either from the primary disease or from a complication.

In this article only those patients who had suffered from bacterial endocarditis were chosen for study. The author points out that there is no very clear agreement as to what is meant by a vegetation. Many authors state or imply

[1] *The Journal of Laboratory and Clinical Medicine*, December, 1946.

that the process of thrombosis is involved, but if the meaning is taken to be the lesion which is characteristic of rheumatic disease, this is misleading. Moore lays down that a vegetation in either acute or subacute bacterial endocarditis has three definite layers, a central mass of necrotic and largely acellular debris, a more peripheral layer of bacteria, and an outer layer of delicate fibrillary material. Collagen fibrils may be demonstrated, but fibrin, though present, is not conspicuous. In the innermost layer it is necrosis that is the most striking feature, and this core of the vegetation makes up over three-quarters of the total bulk. Detailed descriptions of the lesions are given, with illustrations of the histological pictures. On the basis of his observations the author concludes that healing in bacterial endocarditis consists of five processes—covering of the exposed part of the vegetation with fibrous tissue, invasion and phagocytosis of the bacterial colonies, the later calcification of these colonies, and of the central core, and the covering with endothelium of the crevices in the vegetation. If the surrounding fibrous tissue at the edges is not degenerate and if the blood supply is favourable, active fibroblasts will push along the superficial layer of fibrin. The central core, being poor in fibrin and characteristically necrotic, will be one of the slowest parts to show healing. Comparison of the findings in patients who have been treated with penicillin and those who have not, showed that a similar process was discernible in all, but was much more striking in those who had received penicillin. Such a healing process, the author proceeds, may be conceivably stimulated or favoured by four means: by control of bacterial growth, by helping phagocytosis, by promoting the growth of granulation tissue, and by helping the dispersal of necrotic tissue. In penicillin the first requirement is met at least better than by methods previously used. The other requirements leave us without any very definite suggestions. But Moore is not pessimistic altogether; he suggests that possibly some means of encouraging phagocytosis may yet be found. Perhaps we may even yet see some of the ideas of Wright take more definite therapeutic shape. Then with regard to the stimulation of granulation tissue, to gain fibrous tissue adequate supplies of protein and vitamin *C* are needed; Moore suggests that the level of albumin in the serum and the adequacy of the supply of vitamin *C* might be more carefully considered. Whether it is desirable to attempt to encourage calcification by active dietary measures is another matter. In any case one wonders if there really are any active measures feasible. Finally the author cannot see that his studies afford any good reason for using an anti-coagulant such as heparin. However, the value of this agent is perhaps to discourage the secondary thromboses and embolic phenomena that add to the perils of the disease. He postulates that there are two parts in the pathological process involved in the production of the valvular lesions, a non-bacterial endocarditis either rheumatic or thrombotic, and a bacterial superadded infection. This is in accordance with most traditional teaching; so, too, is the explanation of the finding of huge calcified lesions, especially on persons who have shown signs of cardiac inadequacy, by assuming that a mild bacterial endocarditis has progressed to recovery, after being engrafted on previous rheumatic lesions. Certainly it does us good to think again about the effects of treatment in terms of morbid anatomy, without which the concepts of medicine are apt to become uncontrolled.

## SUBCLINICAL POLIOMYELITIS.

BEFORE the modern knowledge about poliomyelitis had been gained, and at a time when virus infections had not assumed an established place in medicine, it was firmly held by many experienced observers that larval or subclinical forms of poliomyelitis were very common. The curious epidemiological features of the disease and the clinical data collected during epidemics all pointed to the same hypothesis. Now it is possible to assemble a great deal more information, and to make an approximate estimation of the frequency of the disease in the community. It would indeed be strange if it were not the lot of each successive generation to acquire its own immunity to this infection, and it is probable that this occurs by the passage of the virus, followed by a very mild systemic reaction, not sufficient to cause definite illness nor to leave any of the unfortunate sequels in the nervous system, but nevertheless competent to produce or perhaps enhance immunity.

A research designed to discover just how widespread is subclinical infection of contacts of the disease has been carried out by A. E. Casey, W. I. Fishbein, I. Abrams and H. N. Bundesen.[1] During the summer of 1945 a coordinated study of poliomyelitis was carried out in Chicago. The contact children were followed up, and observation was made of their clinical condition, with careful record of the body temperature and in as many cases as possible examination of the spinal fluid. After the completion of the investigation the figures and details were analysed by other observers so as to eliminate any question of bias. Studies were made of intimate contacts, of non-contacts living close to the affected children, and also of children of the same age living in another part of the city. It was found that better accounts of the illnesses observed in the children were given by physicians than by nurses. This would be expected; it emphasizes the need for care in collecting data in such inquiries. Instances occurred in which non-paralytic poliomyelitis was reported, but on careful scrutiny the condition was thought to be encephalitis due to mumps. In no case had a child, admitted for the purposes of the investigation, given a history of a prior disease compatible with poliomyelitis. Swabbings were made of the nose and throat, specimens of stool were collected, and the axillary temperature was taken, this method being found to be more reliable and to give more constant readings than the rectal readings. Daily temperature readings on normal controls were used to correct the other findings. Among 22 children who had been in contact with patients with poliomyelitis during its infectious period, 14 showed a fever compatible with poliomyelitis between seven and twenty-five days after exposure. No such disturbance was observed among the non-contacts living close to the affected children. Three out of 37 other controls had fevers which might have been compatible with poliomyelitis, though not definite enough for a diagnosis to be made. In only one of the suspicious illnesses did paralytic poliomyelitis appear; in two others a diagnosis of non-paralytic poliomyelitis was made; in the others there were no signs of diagnostic importance. Examination of the spinal fluid showed increase in cells in one instance and in the protein in another, the number of tests carried out on other children being too small and the delay before the specimens of fluid could be obtained being too long after exposure to infection for the results to be trustworthy.

The authors emphasize that this series is small, and the whole investigation is preliminary, but they believe that the rigid selection of material gives their results some value. Shortage of laboratory animals has prevented them from presenting the results of their virus studies up to the present. This research employs four methods of investigation: careful study of the history and physical state of contacts, the daily determination of temperature, the examination of the spinal fluid, and the determination of the presence of virus in the secretions of the nose, throat and mouth and in the stools. To carry out such a programme in a large series would be ambitious, but it is to be hoped that further studies will be made. These authors believe that they have shown that there is evidence in favour of the belief that poliomyelitis is a disease which is widely communicable, but that about 98% of the attacks are exceedingly mild and leave no residual paralysis. They suggest that to isolate the most intimate contacts of children who have contracted the disease and to make careful studies of their axillary temperatures and to collect stools from those who had definite fever would provide the material for a research which would be useful and informative and not too complex.

[1] *American Journal of Diseases of Children*, December, 1946.

# Abstracts from Medical Literature.

## PÆDIATRICS.

### Penicillin and Congenital Syphilis.

R. V. PLATON, ALLAN J. HILL, NORMAN R. INGRAHAM, MARY S. GOODWIN, ERLE E. WILKINSON AND ARILD E. HANSEN (*American Journal of Diseases of Children*, December, 1946) analyse the results of treatment with penicillin of 191 children under the age of two years who were suffering from congenital syphilis. The authors consider that, though the best method for its use has not been defined, penicillin appears to be the best single agent for the treatment of infantile congenital syphilis. The total dosages employed for effective treatment have varied from 20,000 up to 150,000 units per kilogram of body weight. It is recommended that dosage schedules in excess of 40,000 units per kilogram be used and that treatment continue for sixty or more equal intramuscular injections given at intervals of three hours. Schedules comprising 80,000 or more units per kilogram, divided into 120 equal intramuscular injections given every three hours night and day for a period of fifteen days, have not yet been evaluated. Reactions to therapy have appeared unimportant. Only a relatively small number of infants manifest negative findings on a serological test during or soon after a single course of penicillin; the number showing negative findings increases with each month of observation for at least eighteen months. Careful examinations at regular intervals are necessary to recognize the small number of relapses (determined on either clinical or serological examination) in which the patients require additional therapy. Of the infants treated in this series, twenty-four died; all relevant facts are tabulated in the report and the circumstances of death are analysed. The deaths did not appear to be related to the use of penicillin or to therapeutic shock. A footnote to the article based on further observations recommends a total dosage of at least 100,000 units of penicillin per kilogram of body weight, divided into approximately one hundred and twenty equal intramuscular injections, given at intervals of no longer than three hours "around the clock" for a period of twelve to fifteen days.

### Carcinoma of the Adrenal Cortex.

ALBERT E. GOLDSTEIN, SEYMOUR W. RUBIN AND JOHN A. ASKIN (*American Journal of Diseases of Children*, November, 1946) have reported a case of adrenal carcinoma with the classic adrenogenital syndrome—the eighth case to be reported in the world's literature in which there was a successful operation on a child for this condition. The child, who was aged eight months, is the youngest patient in whom this operation has been successful. This is the first instance in which biopsy of the adrenal glands was utilized to substantiate a pathological diagnosis, a procedure which led to early surgical intervention and cure; this procedure is therefore recommended. The authors state that those patients who manifest abnormal androgenic activity have a great chance of survival; those who manifest abnormal metabolic hormonal activity have a lesser chance of survival, and their course usually terminates with a manifestation of adrenal insufficiency. An approach to the adrenal gland through the lumbar region proved satisfactory in the case of the patient discussed. The world's literature has been searched for cases of adrenal carcinoma in which there was the adrenogenital syndrome; fifty-four cases have been found and analysed, a summary of the history of each patient being included in the paper.

### Hypertrophic Pyloric Stenosis.

GILBERT B. FORBES AND JANE A. ERGANIAN (*American Journal of Diseases of Children*, December, 1946) describe their experiences in treating alkalosis due to pyloric obstruction by the parenteral administration of ammonium chloride. In the case of nine infants with congenital hypertrophic pyloric stenosis, alkalosis of the metabolic type was successfully ameliorated by the intravenous or subcutaneous injection of a solution of ammonium chloride in one-sixth molar concentration in isotonic solution of sodium chloride or Ringer's solution. Toxic effects were observed in only one patient, in whom the material was injected intravenously at an excessively rapid rate. The authors consider that, correctly used, this agent will be of value in the treatment of patients with extreme alkalosis and in the more rapid preparation of patients for operation. Preliminary experiments on dogs to determine the most suitable route for administration are described; subcutaneous injection is favoured. Four important considerations in the clinical use of ammonium chloride are given: (i) it should be combined with some solution containing sodium if lasting benefit is to be obtained and further dehydration avoided; (ii) the other important aspects of pre-operative treatment should not be overlooked; (iii) the treatment should be limited, except in certain emergencies, to those patients for whom accurate chemical data are available; (iv) the solution should be given subcutaneously whenever the patient's condition permits; intravenous injection, if unavoidable, should be made slowly.

### Staphylococcal Pneumonia.

SIDNEY BLUMENTHAL AND HAROLD NEUHOF (*American Journal of Diseases of Children*, December, 1946) discuss the surgical aspects of staphylococcal (suppurative) pneumonia in infancy and childhood based on observations of forty proved cases of bronchogenous infection. A concept of the pathogenesis of the disease is proposed with its classification into a number of types. The pathological and clinical manifestations of the various types, with illustrative case reports, are described. Emphasis is placed on certain radiographic features which when present are deemed to be characteristic or at times pathognomonic of the disease. The authors state that, until the advent of penicillin, staphylococcal pneumonia, often characterized by high mortality, was in the group of self-limited diseases influenced slightly, if at all, by drug therapy; evidence now points to penicillin as a drug which at times exerts a specific effect on the disease. The ætiological importance of a particular staphylococcus in pneumonias of early infancy and the likelihood that any pneumonic lesion in a young infant is of staphylococcal origin are emphasized. The clinical manifestations are stressed, so that the diagnosis can be suspected early in the disease, the time at which penicillin therapy is most effective. The surgical complications of the disease are described. Timely surgical intervention for pleural complications can be life-saving, particularly where there is rapid accumulation of pus, with or without the addition of air. Closed drainage is advocated for such patients and temporizing methods are deplored. Attempts at sterilization of the pleural infection by penicillin are not advocated, because only unsatisfactory results have been noted in limited personal experiences. If local administration of penicillin for empyema is to be employed, it should be reserved for patients who are not suffering from severe toxæmia and/or the mechanical effects of mediastinal displacement. Staphylococcal pulmonary abscess, to be anticipated rarely in the era of penicillin, is amenable to cure by correctly planned surgical drainage through viscero-parietal pleural adhesions.

### Varicella Neonatorum.

P. F. LUCCHESI, A. C. LA BOCCETTA AND A. R. PEALE (*American Journal of Diseases of Children*, January, 1947) report two cases of varicella occurring in newly born children. The exanthem appeared on one child on the eighth day, and on the other child (a premature baby) on the ninth day after birth. The disease clinically was not unlike that which appears in older patients. The premature child died; examination *post mortem* revealed widely disseminated visceral lesions in the lungs, liver and gastro-intestinal tract, associated with the usual skin changes. These lesions were characterized by multiple areas of focal necrosis and by intranuclear inclusions. No specific treatment for varicella in the newborn is suggested, but the authors consider that it is advisable to administer convalescent serum prophylactically to the baby when it is known or suspected that the mother has recently been exposed or when she contracts varicella after parturition. The mothers of both the children discussed were suffering from the disease; the authors discuss the question of when the infection of the children took place in these two cases and in other cases reported in the literature.

### The Incidence of Allergy in Children with Rheumatic Fever.

MARJORIE RITTWAGEN, FRANCIS J. ROMANO AND MORTON PAUL SVIGALS (*Archives of Pediatrics*, December, 1946) investigated the occurrence of allergic conditions in the personal and family history of 100 children suffering from rheumatic fever and compared them with a similar group of children who were not suffering from rheumatic fever. Only such pathological allergies as hay fever, bronchial asthma, food sensitivity and vasomotor rhinitis were included in the criteria of allergy. Of the rheumatic fever patients, 65% gave a positive history of allergy either in themselves or in their immediate families; only 18% of the control group gave such a history. The incidence of allergic diseases in the families of rheumatic children was also much greater than in the general population. A study of the familial distribution of allergy in rheumatic patients is also made and the hereditary factors concerned in allergy are discussed. The

authors state that the exact significance of their findings with respect to the ætiology of rheumatic fever cannot be evaluated at the present time, but make several conjectures.

## ORTHOPÆDIC SURGERY.

### Arthroplasty of the Temporo-Mandibular Joint in Children.

G. W. N. EGGERS (*The Journal of Bone and Joint Surgery*, July, 1946) states that ankylosis frequently recurs after arthroplasty of the temporo-mandibular joint in children. He believes that mandibular pressure on the joint is great in children, and is probably a factor in destroying the interposed fascia, which is usually inserted to secure a false joint. He decided to try a more lasting material, and used tantalum foil in one case. One piece of foil, cut considerably larger than the denuded bone surface, was folded and placed over the base of the skull after the joint had been excised. A second piece of foil, similarly cut and folded, was placed over the mandibular end of the excised joint. Thus four layers of tantalum foil were interposed between the bone surfaces. The author points out that the foil cannot be fastened with sutures, but the mandibular pressure packs it firmly against the denuded bone and creates an area which discourages osteogenesis and the recurrence of ankylosis. The author states that the patient so treated showed satisfactory function of the jaw thirteen months after the operation.

### The Management of Penetrating Wounds of the Knee Joint.

OSCAR P. HAMPTON, JUNIOR (*The Journal of Bone and Joint Surgery*, October, 1946) has studied closely nineteen patients suffering from wounds of the knee joint, with potential, early or late sepsis within the joints. The author compiled data on these patients. Approximately 35 other patients were also observed and studied, but no data were gathered. Both groups of patients had been wounded; they had received initial surgical treatment in forward hospitals a few days or weeks before and had been transferred to base hospitals. The author did not observe any sepsis of the knee joints in those patients who had been treated by complete initial surgery, closure of the synovial membrane or capsule of the joint and adequate immobilization. An aggressive surgical regimen under close observation was instituted for each of the nineteen patients suffering from sepsis of the knee joint. Arthrotomy was performed in all cases. Devitalized areas of articular cartilage, whether due to missiles or to infection, were found and removed. Debris, foreign material, blood clot and pus were removed by thorough irrigation. If the menisci were found to be devitalized and friable, they were removed. Compound comminuted fractures of the patella were noted as the source of sepsis in several instances and the patella was removed. Limb-endangering sepsis in three knee joints was eradicated by resection of the joint. The synovial membrane was sutured in all cases and penicillin instilled into the joint cavity. After surgical treatment each joint was immobilized by a single hip spica or by a Tobruk splint, until it was certain that all danger of a flare-up of sepsis was past and that the wounds were healing. Then the joints were mobilized. Penicillin in doses of 25,000 units intramuscularly every three hours was employed until the danger of continuing sepsis was past, and usually until all the wounds had healed. Aspiration, irrigation and reinstillation of penicillin (1,000 units per millilitre) were also carried out at intervals of twenty-four or forty-eight hours for about one week. Very little fluid reaccumulated, however, and the attempts at aspiration were usually unsuccessful. Blood transfusions were given in amounts which maintained a hæmatocrit level of at least 40% of red cells. The author states that observation made in these cases led to the standardization of a plan for the management of all knee joint wounds due to shell fragments or bullets. Thorough excision was the most important factor in obtaining successful results. At the time of the initial surgical treatment, the preferred plan of management for penetrating wounds of the knee joint, regardless of the time interval between receiving the wound and operation, should include (*a*) arthrotomy, (*b*) excision of the devitalized tissue, including areas of destroyed articular cartilage, (*c*) cleansing of the blood clot and debris from the joint, (*d*) suture of the synovial membrane or capsule, and (*e*) immobilization of the limb in a hip spica or a Tobruk splint. At the time of reparative surgery, preferably from five to ten days after the intraarticular surgery, the skin should be sutured. Immobilization should be discontinued as soon as the danger of sepsis is past and the skin has healed, or is healing. Active and passive mobilization should then be instituted. The author believes that in cases of suppurative arthritis of the knee joint, potential or established, the same surgical plan is indicated. Continuous drainage of fluid from the knee joint is not adequate surgical treatment in cases of suppurative arthritis; moreover, it is not indicated. Systemic administration of penicillin (at present the best available antibacterial method), supplemented by the local instillation of penicillin into the joint, should be employed in the treatment of patients with wounds and suppurative arthritis of the knee joint, in order to prevent invasive infection of the living tissue. The author considers that in the light of the problem and the hazards of knee-joint sepsis, knee-joint resection has a definite, but fortunately a limited, application in the management of severe wounds and sepsis of the knee joint in military surgery.

### Complications of Fractures of the Neck of the Femur.

H. B. BOYD AND I. L. GEORGE (*The Journal of Bone and Joint Surgery*, January, 1947) present the complications and end results of internal fixation of 300 fractures of the neck of the femur. Smith-Petersen nails were used in 285 cases and Knowles pins in 15. All impacted fractures and fractures of the base of the neck were excluded, only Pauwel type two and three fractures being included. In all cases one year or longer had elapsed since operation. There were 28 deaths following the 300 operations, 21 of these having occurred within the first four weeks. The authors believe that this represents the true operative mortality. All the patients who died were over sixty years of age, and the mortality increased with age until it reached 25% in the ninth and tenth decades. Seven deaths were recorded from four weeks to six months after operation. The authors consider that undoubtedly a number of other patients whom they were unable to trace died within the period of four weeks to six months after operation. The post-operative follow-up period of the 131 fracture patients was less than one year and was therefore not considered in determining the end results. One hundred and forty-one patients with fractures were followed for one year or longer, and among these the authors found that bony union took place in 86·5%; union failed to occur in the remaining 13·5%. The authors consider that frequently mistakes will be made if an endeavous is made to determine the presence of bony union under one year. In ten of the nineteen ununited fractures, poor reduction, inadequate mechanical fixation, premature removal of the nail, or a combination of these factors, contributed to the non-union. The remaining nine ununited fractures followed satisfactory reduction and nailing; two of these fractures were in patients with Paget's disease. Aseptic necrosis and arthritic changes in the hip each occurred in 33·6% of cases. The authors draw the following conclusions following 300 cases of fracture of the neck of femur, among which 131 patients were followed for less than one year and were not considered in regard to the prognosis. A poor end result occurred in 28·1% of cases (these including ununited fractures and those in which severe arthritic changes occurred); a fair end result occurred in 19%; and a good end result in 43·6% of cases. The data presented indicate that the total number of patients with severe arthritic changes in the hip will exceed the number with ununited fractures.

### Posterior Approach to the Supero-Lateral Region of the Tibia.

EMANUEL B. KAPLAN (*The Journal of Bone and Joint Surgery*, October, 1946) presents an approach to the upper postero-lateral aspect of the tibia. The author states that a dry, safe field, with exposure of the popliteus, can be obtained if a mid-line longitudinal incision is made, the popliteal region being penetrated on the medial side of the popliteal artery; the medial inferior genicular artery is divided and ligated. The popliteus can be divided to expose the underlying bone. The author claims that with this procedure the exposure of the upper end of the tibia can be accomplished in a few steps. By this approach temporary resection of the fibula is avoided and there is only slight interference with anatomical structures. The author states that the only disadvantage of this approach is the relative depth of the operative field. The position of the fibula, on a more posterior plane than the tibia, restricts somewhat the lateral retraction, but this does not interfere with proper exposure of the field. The angle formed by the tibia and the upper end of the fibula can also be exposed. The author points out that the anterior tibial artery passes through the interosseous membrane at a point varying from one to two inches distal to the apex of the angle formed by the tibia and fibula. This relationship permits ample room for approach to the tibio-fibular angle.

# Bibliography of Scientific and Industrial Reports.[1]

## THE RESULTS OF WAR-TIME RESEARCH.

*During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken from former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.*

*Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) The reports referenced with an E number may be obtained in approved cases without cost on application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.*

*Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.*

PB 40330. FAIR, GORDON M. Disinfection of water. Summary report of the effectiveness of "Bursoline Number 3" tablets in the disinfection of water in canteens. Interim report number 2. (OSRD Committee on Medical Res. Sanitary Eng. Rept. 4.) July, 1944. 9 pp. Price: Microfilm, $1.00; Photostat, $1.00.

This document reports work done under contract number OEMcmr-251. The "Bursoline Number 3 Canteen Water Sterilizing Tablet", manufactured by the Burnham Soluble Iodine Company, has a total iodine concentration of 16·4 milligrammes $I_2$ and produces an average titratable iodine concentration of 7·4 parts per million when a single tablet is added to a quart of water. It dissolves rapidly and produces its maximum iodine concentration in 1·25 minutes at 0° C. and 0·5 minute at 23° C. The tablet remains effective after having been stored in sealed bottles at 140° F. for three months. One tablet per quart is cysticidal at 23° C. in normal waters in five minutes; in moderately polluted, alkaline and turbid waters in ten minutes. The results of cysticidal and bactericidal tests made with "Bursoline" and "Standard Halazone tablets" are tabulated. When canteens are filled by dipping water from polluted sources, the canteen lips and threads are not disinfected even though the water in the canteen itself is rendered safe. "Bursoline Number 3" can be successfully packaged in glass containers.

PB 40328. FAIR, GORDON M. Disinfection of water. Bi-monthly progress report, numbers 1 to 13. October, 1943, to October, 1945. 35 pp. Price: Microfilm, $1.00; Photostat, $3.00.

This document consists of progress reports on work done under contract number OEMcmr-251. The cysticidal efficiency of the following compounds was tested: succinchlorimide, halazone (*p*-sulphone-dichloroamido-benzoic acid), "HTH" (calcium hypoclorite), sapamine (quartenary ammonium compound), "Ceepryn" (cetyl pyridinium chloride), "Roccol" (alkyl-dimethyl benzylammonium chloride), "Triton K-60" (quartenary ammonium compound), "Zephiran" (alkyl-dimethyl benzylammonium chloride), "Aerosol", "Fixanol", hexyl resorcinol, "Burroughs-Wellcome Tablet Number 4" (20 milligrammes KI, 10 milligrammes $KIO_3$, 1·0 milligrammes $KMnO_4$, and 20 milligrammes citric acid), "Bursoline Tablet Number 1, Burnhem Co." (diglycine hydriodide 12 milligrammes, iodine 5·0 milligrammes, disodium-pyrophosphate), "Chlor Dechlor" (Wallace I. Tiernan Co.—halazone powder and coated sodium sulphite tablet), combinations of succinchlorimide and halazone, "Chloramine T and Sodium Sulphite Dechlorinating Tablets" (Lilly Co.), "Activated Succinchlorimide" (Lambert Co.), lithium hypochlorite, "Activated Chloramines" (University of Wisconsin), iodo-succinimide (Wallace I. Tiernan Co.), "HTH" and "Fixanol", halazone and "Fixanol", iodine and "Fixanol", "Bursoline Tablet Number 3", succinimide periodide (Wallace and Tiernan), "Globaline tablets", "Alure", and triglycine hydroperiodide. The following bacteria were used in the tests: *Entamœba histolytica, Escherichia coli, Eberthella typhi.*

PB 40329. FAIR, GORDON M. Chlorination of cysts of *Entamœba histolytica.* Progress report, numbers 1 to 5. December, 1942, to August, 1943. 7 pp. Price: Microfilm, $1.00; Photostat, $1.00.

This document consists of progress report on work done under contract number OEMcmr-251. The cysticidal efficiency of H.T.H. (calcium hypochlorite) with ten and fifteen minute contact periods at temperatures of 18°, 23° and 28° C. was determined. Citric acid was employed for pH control. Other drugs tested were "Halazone" (*p*-sulphone-dichloroamido-benzoic acid) and "Microklene" (mixture containing a detergent and a chloramide compound). "Microklene" was also applied on contaminated cabbage, lettuce and carrots.

PB 40324. FAUST, ERNEST C., AND SMITH, RALPH G. Testing the toxicological and therapeutic action of certain drugs for possible use in treatment of schistosomiasis. Bi-monthly progress report, numbers 1 to 11. February, 1944, to October, 1945. 22 pp. Price: Microfilm, $1.00; Photostat, $2.00.

These reports deal with methods to infect monkeys, rabbits, rats, hamsters *et cetera* with *Schistosoma mansoni* for testing the therapeutic value of antimonials. Preliminary toxicity tests of antimony in rats were made with antimony and potassium tartrate, "Anthiomaline", "Fouadin" and "Neostibosan". Therapeutic results of treatment with these drugs are described. Furthermore, attempts were made to develop methods for the quantitative determination of organic antimonials, including "Anthiomaline" in urine and tissue. The polarographic determination of trivalent antimony in urine and blood is described.

PB 40322. FAVOUR, CUTTING B. Preservatives for biological products, especially blood products. Progress report, numbers 1 to 6. September, 1944, to July, 1945. 5 pp. Price: Microfilm, $1.00; Photostat, $1.00.

Carboxymethoxylamine hemihydrochloride, toxicity of which was studied in mice and men, is most useful in concentrated protein solutions where other preservatives are ineffective. One hundred milligrammes *per centum* of the compound are sufficient to prevent bacterial growth of inocula up to 2,000 organisms per millilitre and to kill such an inoculum at room temperature or 37° C. within one to fourteen days depending on the bacterial strain. Dosage and preparation of the solutions, media and bacteria used in the various experiments are described in detail in these reports. The work reported was done under contract number OEMcmr-453.

PB 37061. U.S. WAR DEPARTMENT. Complications of blood transfusion. (Technical Bulletin MED 204.) October, 1945. 14 pp. Price: Microfilm, $1.00; Photostat, $1.00.

The complications of whole blood transfusion may be outlined as follows: 1. Immediate untoward reactions, (*A*) hæmolytic, (i) intravascular hæmolysis of incompatible donor cells, (*a*) intergroup, (*b*) intragroup, (ii) intravascular hæmolysis of recipient's cells, (iii) intravascular hæmolysis of compatible donor cells, (iv) transfusion of hæmolysed blood, (*B*) pyrogenic, (*C*) allergic, (*D*) circulatory overload, (*E*) embolic. 2. (*A*) Infectious hepatitis, (*B*) malaria, (*C*) syphilis, (*D*) other diseases. Of all the complications listed above the hæmolytic reaction resulting from transfusion of incompatible cells is the most important, because it often results in a fatality. The pyrogenic reaction is next in importance because of its frequent occurrence. The bulletin gives detailed directions for diagnosing the clinical manifestations and for preventing contraindicated transfusions.

PB 23055. WEST, HOWARD, *et alii.* Card sorting test for demonstration of anoxia. Some factors influencing scores made on the Seattle card sorting test. (Bur. of Medicine and Surgery Project X-283, Report 1.) June, 1944. 16 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The results of this sample field check of the card sorting test suggest the need for a further study to establish the validity and reliability of this test with large numbers of subjects. As indicated by a limited number of subjects using a small sample technique, the card sorting test was found to be unsuitable for use in demonstrating the effects of anoxia, because (*a*) the effect of learning, which is characteristic of successive administrations of such material,

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

has important influence on the results; (*b*) the results under anoxic conditions are influenced by compensatory factors; (*c*) the test is relatively difficult to administer and the bulk and number of boxes necessary for routine use would make for increased difficulties in already crowded chambers. A bibliography, four tables and two graphs are attached.

PB 37051. U.S. WAR DEPARTMENT. Oxygen therapy outfit, hose line assembly, 20 dual outlets, complete. Care and maintenance. (Technical Manual 8-637.) December, 1944. 9 pp. Price: Microfilm, $1.00; Photostat, $1.00.

The "oxygen therapy outfit, hose line assembly, 20 dual outlets, complete", is designed primarily for the administration of oxygen to a large number of patients at one time. These instructions contain information on the operation and maintenance of the equipment as well as a description of the major units and their functions. Two drawings of the equipment are included.

PB 37060. U.S. WAR DEPARTMENT. Treatment programme for psychiatric patients in station and general hospitals. (Technical Bulletin MED 84.) August, 1944. 7 pp. Price: Microfilm, $1.00; Photostat, $1.00.

The acute needs for manpower make it imperative that every treatment method available in station and general hospitals for neuropsychiatric patients be utilized to the maximum. The aim is to salvage every possible soldier for further duty. Directions are given as to how to house psychotic and psychoneurotic patients and what kind of personnel and equipment to use. In the section "Treatment Methods" the following topics are discussed: psychotherapy, sedation combined with psychotherapy, occupational-recreational-educational activities, shock therapy, hydrotherapy and drug therapy.

PB 40321. FENDER, FREDERICK A. MRPD 91: Electroencephalography. Progress report, Numbers 1-7. August, 1942, to June, 1943. 14 pp. Price: Microfilm, $1.00; Photostat, $1.00.

This document consists of seven progress reports under contract number OEMcmr-163, MRPD 91. The first two describe in great detail procedure, equipment and scoring. Data obtained from fifteen subjects on the tolerance to alcohol and anoxia, skill, rate of learning and peculiarities of the electroencephalograms are tabulated. In the course of investigation the actual conduct of the tests was repeatedly revised. Alcohol was replaced with "Sodium amytal". According to the last report electroencephalograms on 650 candidates for naval aviation were secured. These records are in the process of being read and rated according to the scheme of Dr. Hallowell Davis. The project awaits the records which these individuals achieve in training and operations. When they become available it will be possible to determine any correlation of performance with electroencephalographic record.

PB 40317. FIESER, LOUIS F. Synthesis of *E* progress report, Numbers 1-12. August, 1942, to May, 1944. 15 pp. Price: Microfilm, $1.00; Photostat, $1.00.

This document consists of brief progress reports on the synthesis of vitamin *E* for the period of June 25, 1942, to May 16, 1944. The work was performed under contract number OEMcmr-168. Crystalline methyl 3, 12-diacetoxy desoxycholate was prepared as starting material. Stepwise oxidative degradation was performed by applying (1) the Ruzicka oxidation of the side chain and (2) the oxidation of acid peroxides. Among other reactions tried were: attempted $C_{11}$-substitution of $C_{12}$-ketones, preparation of methyl diacetyl desoxycholate and its derivatives, oxidation of methyldibenzoyl desoxycholate, promination of $\Delta^{11,12}$-lithocholenic acid, preparation of 12-keto-cyanohydrin, extraction of cortical hormone metabolites as acetates, and side chain degradation through the diene.

PB 40343. MOORE, J. E., AND EAGLE, HARRY. Studies on the serologic pattern of syphilitic and non-syphilitic human sera and animal sera. Monthly progress report, Numbers 1-3. January to June, 1944. 5 pp. Price: Microfilm, $1.00; Photostat, $1.00.

This project was sponsored by the Committee on Medical Research of the Office of Scientific Research and Development. A preliminary analysis of 473 cases showed the following results: (1) In general in syphilis, either congenital or acquired, sera containing small amounts of reagin give a flocculation fibre which is slightly greater than the complement-fixation titre. In sera containing large amounts of reagin the complement-fixation titre is higher than that of the flocculation test. In false positive sera the same would seem to hold true. In those with low titres the flocculation test is more sensitive, in those with high titres the complement-fixation test is more sensitive. (2) False positive sera do not react with non-specific antigens. Non-specific reactions occur, if anything, more frequently with syphilitic sera. (3) Kahn's "verification" test, the Rein special technique, and the Eagle spirochætal complement-fixation test behaved in similar fashion. In each there was a close correlation between titre and type of reaction. Sera with low titres gave a biological type of reaction and those with high titres gave a syphilitic type of reaction regardless of whether the serum was from a syphilitic patient or from a person with a false positive reaction. It is concluded that the diagnosis of syphilis or false positive reaction is a function of the clinician rather than of the serologist.

PB 40339. MORGAN, DAVID W. Analysis of one hundred psychiatric casualties and one hundred apparently normal soldiers. Bi-monthly progress report, Numbers 1-5. October, 1942, to May, 1943. 5 pp. Price: Microfilm, $1.00; Photostat, $1.00.

The work of the investigators consisted chiefly of follow-up studies of the individual cases, review of the literature on psychiatry and neurology as it applies to war, completing clinical records, psychological testing (Rorschach), and statistical analyses. Two papers were written as a result of the studies indicated: "An Analysis of One Hundred Army Psychiatric Cases and One Hundred Enlisted Men" and "A Note Regarding the 'M-Score' of Penrose and Myers". This work was done under OEMcmr 65, Supplement No. 1, with the Committee on Medical Research of the Office of Scientific Research and Development.

PB 20450. BICKENBACH. *Zusammenfassung der Ergebnisse der Oberflächenspannungs- und Viskositäts-untersuchungen am Serum in vitro.* (Summary of the results of a study on surface tension and viscosity in serum *in vitro*.) (ALSOS Mission, RFR 472.) No date. 7 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Pure phosgene was passed through untreated serum, leading to measurably increased content of $C_1$, to increased viscosity of the serum, and to a slightly lowered surface. In German.

PB 23274. INSTITUT FÜR ERNÄHRUNG UND HEILPFLANZENKUNDE, DACHAU. *Die Vitaminversorgung der Truppen.* (Vitamin supply for soldiers.) September, 1942. 122 pp. Price: Microfilm, $1.50; Photastat, $9.00.

This is a series of lectures delivered in the above institution dealing with (1) the experiences on the vitamin supply of soldiers in the field; (2) the rose hip as a source of high-grade vitamins, its collecting, working up and use; (3) administrative measures of the army to guarantee vitamin supply and preservation; (4) the vitamin supply of the service forces; (5) vitamin control in the army; (6) new ways for the disclosure of natural vitamin *C* sources; (7) obtaining vitamin *C* from gladiolus; and (8) the horticultural grounds of the German Experimental Station for Nutrition and Feeding at Dachau. In German.

PB 20479. KREIDEWEISS. *Vorläufige Mitteilung über die Einwirkung von Spirochæte pallida auf maligne Tumoren.* (Effect of *Spirochæta pallida* on malignant tumours; preliminary report.) (*Reichsforschungsrat.*) (ALSOS Mission, RFR 106.) 27 pp. Price: Microfilm, 50c.; Photostat, $2.00.

After making a survey of the literature on the frequent association of malignant tumours and syphilis, in the course of which he noted the many contradictory opinions expressed regarding the true relationship between the causative agent of syphilis and the growth of tumour cells, the author, believing that this relationship is an antagonistic one, injected syphilitic serum into seven patients suffering from various types of malignant tumours (skin cancer, rodent ulcer, metastatic mammary cancer, fusocellular and globocellular sarcoma) and observed the results. In all seven cases the tumours either rapidly diminished in size or disappeared completely. Three of the open types of cancer healed completely, with cicatrization. The antagonistic action of *Spirochæta pallida* seems to depend, the author says, on the stage of incubation, that is, as soon as the spirochætes disappear from the system, and immunity sets in, no further check on the growth of the tumour cells is noted. He suggests that, if the active agent of *Spirochæta pallida* could be isolated in such a way as to preserve properties which the living organism possesses, it might be possible to find a way of effectively destroying malignant tumour cells. An annotated bibliography accompanies the article. In German.

PB 23275. SCHENCK, E. G. *Grundlagen und Vorschriften für die Regelung der Krankenernährung im Kriege.* (Basic principles and instructions for regulating the nutrition of the sick during wartime.) April, 1940. 94 pp. Price: Microfilm, $1.00; Photostat, $7.00.

This is a collection of all orders and instructions dealing with the nutrition of the sick within the German system of food rationing after the outbreak of the war. It is divided into (1) general principles, (2) organization of the nutrition of the sick during wartime, (A) procedure on behalf of individuals, (B) regulation of the nutrition in hospitals, convalescent homes and health resorts, (3) general directions for the allotment of food. They are based on medical prescriptions and permits issued by the proper food authorities. Special attention is given to various diseases and special conditions. In German.

# British Medical Association News.

## SCIENTIFIC.

A MEETING of the Section of Pædiatrics of the New South Wales Branch of the British Medical Association was held on October 30, 1946, at the Royal Alexandra Hospital for Children. Part of this report appeared in the issue of May 17, 1947.

### Patent Ductus Arteriosus.

DR. T. Y. NELSON said that he had a case of patent *ductus arteriosus* to present to the meeting; but before showing the child he would outline some of the problems which were met in the treatment of these cases. The first difficulty was in the diagnosis, and although the signs in a typical case presented a definite picture which could be recognized with certainty, there were other cases in which the diagnosis was not straightforward. The typical signs described were a characteristic murmur, a palpable thrill and X-ray evidence of an enlarged left ventricle and pulmonary congestion. Probably the most important clinical sign was a lowering of the diastolic blood pressure, which would sometimes be apparent only after exercise. If the diagnosis was established, the next problem was to decide which patients should be submitted to operation and at what age this should be done. There was inevitably considerable conservatism in the early stages of development of this operation, and it was thought that signs of cardiac failure should be present before operation was justified. But there was now a tendency to enlarge the scope of the operation, and some observers considered that if the diagnosis could be made with certainty, all patients should be subjected to operation in childhood. Bacterial endocarditis had been recognized as an absolute indication for operation, and in spite of the beneficial effects of penicillin therapy, this statement still held. Dr. Nelson thought that the decision whether operation should be performed must be affected by the mortality rate and by the improvement in surgical technique which would guarantee successful closure of the duct in all cases. As no series of statistical significance had been published in Australian literature, it was necessary to consult the literature of other countries to assess the risks of the operation. Shapiro had made an important survey of the available cases in American literature, and found that 140 patients had been operated on by 25 different surgical teams. Although three cases were unrecorded, the operative mortality of the whole series was in the region of 8%. As this series included the earlier patients who had been treated in each clinic, it should be capable of considerable improvement and the ultimate figures should be considerably lower. In successful cases the results of operation were dramatic. Dr. Nelson demonstrated X-ray films of a patient who had been subjected to operation; the films showed the reduction in the size of the heart shadow two weeks after operation.

Dr. Nelson then showed a girl, aged three and a half years, showing characteristic signs of the abnormality, and asked for comments on whether the child should be submitted to operation. He said that the condition had been recognized at the age of two and a half years, when the child was admitted to hospital for an acute febrile illness. The murmur was of a characteristic harsh, continuous type, the X-ray film showed enlargement of the heart, and the blood pressure recorded on several occasions varied after exercise from 105 to 120 millimetres of mercury (systolic) and from 40 to 20 millimetres (diastolic). The only abnormality that the mother had noticed was that the child tired easily and did not want to play about as much as a normal child. There was no obvious retardation of growth. Dr. Laurence Hughes had examined the patient and agreed with the diagnosis.

DR. LINDSAY DEY said that he had had a long discussion with Dr. B. T. Edye about the indications for operation. Dr. Edye agreed with Dr. Nelson that, apart from endocarditis, physical retardation was the best indication, and had said how pleasing it was to see the improvement in these children after operation. Dr. Dey further referred to the necessity for a rational outlook on the question. He knew of a woman, aged thirty-five years, who had borne at least two children without trouble, and who desired operation for purposes of increased capability of indulging in sport. It seemed that this was a case in which operation was a risk that was hardly indicated.

DR. CLIFTON WALKER said that Dr. Nelson had quoted an authority as stating that all patients with patent *ductus arteriosus* should be subjected to operation. Dr. Walker presumed that uncomplicated cases were meant, as the presence of an open *ductus arteriosus* was essential as a compensatory by-pass when certain congenital anomalies of the heart and great vessels were present. Dr. Walker asked Dr. Nelson whether he had any ideas on the mechanism of closure of the *ductus arteriosus*. If this was known, it might be possible to decide the reason for the failure to close in certain cases.

DR. R. D. K. REYE drew attention to the fourteen cases listed in which the ligated *ductus arteriosus* was said to have become recanalized. He asked whether this referred to a shifting of the ligature and a return of the ductus to the pre-operative state, or whether it was to be inferred that a thrombus had developed in the ligated duct, but that this thrombus had become recanalized. If the latter sequence had occurred, then the flow of blood through the duct must have been appreciably reduced. However, Dr. Reye was interested in this point from the possible light which might be shed on certain cases of abnormal closure of the duct. As Dr. Nelson had indicated in his reply to a previous question, some doubt still existed about the exact mechanism for the closure of this arterio-venous shunt. The question seemed to resolve itself into two components. The first was the mechanism of immediate closure. This was generally assumed to be due to muscular contraction, mainly at the ends of the duct, more particularly at the pulmonary end. The second was the method by which permanent obliteration of the lumen was attained. The normal mechanism concerned in this second event, and occurring during the first few weeks of life, seemed to be one of subendothelial proliferation which took place in certain ridges or cushions already partially developed in the fœtal state. In a few instances, in patients who had died in the first few weeks of life, Dr. Reye had noticed a type of abnormal obliteration of the lumen of the duct by thrombus; in one such case a slight but distinct aneurysmal bulge had been present in the wall of the duct. The duct in these cases showed no signs of becoming obliterated in the usual way, and he wondered whether it was likely, had these patients lived, that recanalization of the thrombus would have occurred. The cases mentioned were not complicated by any other cardiac or vascular defect. In reply to a question from Dr. E. Stephen, Dr. Reye said that he had never seen an example of vegetative endarteritis developing at the site of a patent *ductus arteriosus*, and he felt fairly sure that a search of the hospital autopsy records for the past twenty years would not reveal a single example. All forms of bacterial endocarditis were infrequent at this age period and provided a very low percentage of the autopsies—probably less than 0·5% at a rough estimate.

DR. NORMAN CUNNINGHAM thanked Dr. Nelson for his interesting talk. Dr. Cunningham said that he had seen a number of patients with patent *ductus arteriosus* and proposed to comment on two or three points. The most important of these was the recurrence of the murmur after operation. This was disquieting, and indicated that the operative technique had not been sufficiently developed. One was reminded of the wave of enthusiasm which had swept the country fifteen years earlier when the operation of gastro-enterostomy was introduced for peptic ulcer. There was great enthusiasm; but, as was now known, the operation had been replaced by partial gastrectomy, and surgeons were still spending some of their time in undoing their previous work. Dr. Cunningham said that he had recently had the good fortune to be invited to attend a meeting of the Melbourne Pædiatric Society. At that meeting three patients with patent *ductus arteriosus* were shown by Dr. H. L. Stokes and Dr. C. J. Officer Brown. In two of the cases operation had been performed, and the murmur had recurred in one. In Sydney and Melbourne the murmur had recurred in a number of cases, after disappearing at operation. Dr. Edye had told Dr. Cunningham two days prior to the meeting that for the first time he had reopened a patient's thorax because of the recurrence of a loud murmur; he had found a large aneurysm between the ligature and the aorta; he had done nothing, and one imagined that the prognosis was bad. Dr. Cunningham felt that the over-all mortality rate was between 10% and 15% in Australia, although the total number of cases was too small to analyse. That was not a good rate, and did not include the aneurysm case just mentioned. One had only to see the spectacular results of surgery in patent *ductus arteriosus* to realize that it was the treatment of choice; but until a more certain technique was devised and the surgeon could guarantee that the patient would live longer if subjected to operation than he would otherwise live, Dr. Cunningham could not subject any of his children to operation just because they had a patent *ductus arteriosus*. They would have to have also one of three complications: (i) a failing or enlarging heart, as shown by radiological and clinical examination; (ii) physical or mental retardation

not responding to medical treatment; (iii) infection of the ductus. Dr. Cunningham was interested to learn from Dr. Edgar Stephen's remarks (confirmed by Dr. Reye) that infection of the ductus was rare at the Royal Alexandra Hospital for Children. Dr. Geoffrey Davies, the pathologist at the Royal Prince Alfred Hospital, had said that he had not seen a case in which a patent *ductus arteriosus* was the primary cause of death. That observation gave them food for thought, as did Dr. Reye's remarks; yet they were told in overseas papers that patients with patent *ductus arteriosus* rarely survived their thirties; they must go somewhere to die.

DR. LORIMER DODS said that he had been particularly interested to hear Dr. Cunningham's statement that no example of bacterial endarteritis associated with an uncomplicated patent *ductus arteriosus* had been observed in the post-mortem room at the Royal Prince Alfred Hospital during the past decade; Maude Abbott had stated that such infection occurred in more than 35% of uncomplicated cases of patent *ductus arteriosus* in which the patients had survived for more than five years. Dr. Dods agreed with Dr. Cunningham's criteria for operation, but pointed out that the hazards of subacute bacterial endarteritis and of cardiac failure belonged more to adolescence and early adulthood, while physical impairment belonged to the childhood period and might be reflected in some impairment of school work. It seemed that the signs of physical impairment in a child suffering from uncomplicated patent *ductus arteriosus* were rarely obvious before the fourth birthday; the child shown by Dr. Nelson appeared to be an exception to this generalization.

## MEDICO-POLITICAL.

A MEETING of the Southern District Medical Association, New South Wales, was held on May 4, 1947, "to consider the proposed nationalization of the medical profession in Australia".

A series of fourteen motions were moved by Dr. T. P. Mahon, put to the meeting and carried. It was resolved that the resolutions should be sent to this journal with a request that they be published. The resolutions are as follows:

1. That this meeting of the Southern District Medical Association reaffirms its unequivocal opposition to a whole-time salaried medical service for the Australian community, as it is considered that such a service would be not in the public interest; would inevitably decrease the efficiency of medical practitioners, and be detrimental to the health of the community.

This meeting is further of the opinion:

2. That, in principle, the present doctor-patient relationship is the best known method of ensuring efficient medical attention for all sections of the community, except in so far as the financial burden falls inequitably on those suffering prolonged or serious illness from causes outside their control.
3. That the financial burden can, and should, be shared by all sections of the community.
4. That the financial burden can best be shared by a plan of compulsory national health insurance, due allowance being made for those whose financial circumstances preclude contribution.
5. That the funds of such a compulsory national health insurance plan should be kept completely separate from the general exchequer, and/or funds connected with any other form of social security plan.
6. That the administration of such a plan should be free from departmental or any other method of control which would interfere with the freedom of the profession and the public.
7. That on the administrative body, there should be adequate representation of that part of the medical profession engaging in curative medical practice.
8. That in cooperating with the Government in the provision of decentralized "diagnostic clinics", the profession should strongly oppose the principle of staffing such centres with whole-time salaried medical officers; that part-time salaried officers having the right of private practice would be in the best interests of the public and the profession, bearing in mind that a whole-time salaried service by one section of the profession is likely to be but the first step in complete socialization of medical practice.
9. That in the formulation of a national health insurance plan consideration should be given to a scheme designed to spread the financial burden equitably over the whole community, namely: (*a*) Contributions to be on an agreed upon equitable basis. (*b*) Claims to be based on a certain minimum figure bearing a fixed relationship to annual income, as determined by taxation; for medical expenses falling below that figure, no claim to be allowable; for expenses above that figure, claims to be paid in whole or in part as determined by actuarial investigation. Claims would be settled by a refund of taxation paid. Indigent non-contributors would, of necessity, be a charge on the fund.
10. That immediate steps should be taken to stimulate and if necessary provoke public discussion of: (*a*) The principles involved in and possible implications of nationalization of the medical profession. (*b*) The alternatives advocated by the profession. (*c*) Disabilities complained of by the public at present, as it is considered that only by stimulating the public to ask questions can medical practitioners become efficient publicity agents "at the periphery".
11. That disabilities suffered by the public are almost entirely financial, and where otherwise, would be worsened and not bettered by disturbance of the present doctor-patient relationship.
12. That if funds be not available for such publicity as suggested, immediate steps should be taken to acquire the necessary funds to be placed at the disposal of the Publicity Committee of the Federal Council.
13. That in order to refresh the minds of medical practitioners and help them to enlighton the public, certain motions carried at the meeting of the Federal Council of the British Medical Association in Australia, held in Melbourne, September 25-28, 1944, should be reprinted and distributed to all members of the British Medical Association in Australia. The resolutions laid down, what the meeting considered to be the duties of the Committee of the Federal Council appointed to meet representatives of the Government, and are: Motions 3, 4 and 5 (Clauses B and C) of the series of five motions moved by Dr. H. C. Colville and reproduced in THE MEDICAL JOURNAL OF AUSTRALIA, October 28, 1944, page 464.
14. That further to enlighten the public, a reprint, actual or edited, of the above-mentioned resolutions of Federal Council should be distributed to medical practitioners in a form suitable for display in waiting rooms and that Council should cause to be printed in the daily Press a similar notice by advertisement or otherwise.

## NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the undermentioned medical practitioner has been released from full-time service with His Majesty's Forces and has resumed civil practice as from the date mentioned:

Dr. B. Oxenham, 143, Macquarie Street, Sydney (April 14, 1947).

# Post-Graduate Work.

## MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

### Demonstration at Geelong.

AMONG the country courses to be held during June, 1947, by the Melbourne Permanent Post-Graduate Committee is one at Geelong to be given by Dr. Ewen Downie. In the issue of May 17, 1947, it was incorrectly stated that this demonstration would take place on June 22. The correct date is Wednesday, June 25.

# Correspondence.

## INTERNATIONAL CONGRESS OF MENTAL HEALTH.

SIR: The third congress will be held in London on August 12 to 21, 1948, and will be divided into sections dealing with child psychiatry, medical psychotherapy and mental hygiene. Topics for discussion under these three main headings have already been selected. It is hoped that delegates to the congress will be widely representative, and the organizing

committee is therefore anxious that its plans should be brought to the notice of members of the medical profession as well as to others working in the various fields of mental hygiene.

Anyone who is interested is invited to communicate with the Organizer, International Congress on Mental Health, 39, Queen Anne Street, London, W.1, England.

Yours, etc.,

135, Macquarie Street, Sydney, April 14, 1947. W. S. DAWSON.

# The Royal Australasian College of Surgeons.

## GORDON CRAIG SCHOLARSHIPS.

THE Council of the Royal Australasian College of Surgeons invites applications for the fourth award of Gordon Craig Scholarships.

1. These scholarships are awarded for the purpose of furthering post-graduate education in surgery and surgical research.
2. Favourable consideration will be given to applicants who intend to present for a senior surgical qualification.
3. Applicants must have been qualified for at least two years.
4. Scholarship holders will be required to carry out whole-time duty at a medical school and/or research institute and/or hospital, although this condition may be varied in exceptional cases.
5. The amount of each scholarship will be at the discretion of the College, but the College is prepared to grant up to £400 a year to suitable applicants.
6. Forms of application, together with details of the regulations governing the award of the scholarships, are available from the Secretary of the Royal Australasian College of Surgeons, Spring Street, Melbourne.
7. Applications should be lodged with the Secretary and reach him not later than January 31, 1948.

## EXAMINATIONS FOR FELLOWSHIPS.

THE next meeting of the Court of Examiners for the Primary Examination for Fellowship of the Royal Australasian College of Surgeons will be held in New Zealand probably early in December, 1947.

The next meeting of the Court of Examiners for the Final Examination for Fellowship of the Royal Australasian College of Surgeons will be held at the College in Melbourne in December, 1947.

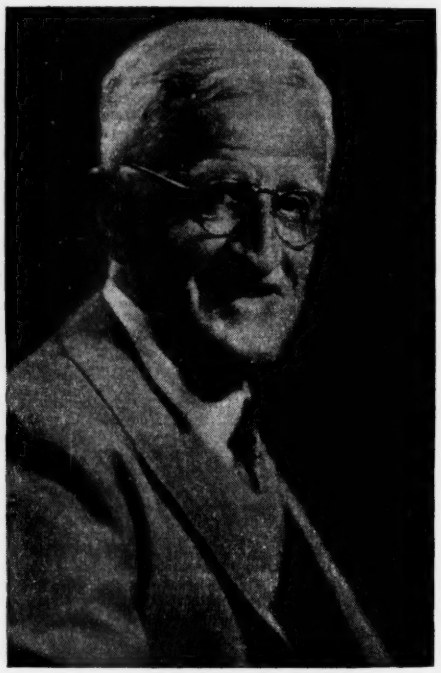
Candidates who desire to present themselves at either of these meetings should apply to the Censor-in-Chief for permission to do so on or before October 1, 1947. The appropriate forms are available from the Secretary of the Royal Australasian College of Surgeons, Spring Street, Melbourne. C.1.

# Obituary.

## RALPH CHARLES BROWN.

WE are indebted to Mr. Balcombe Quick for the following account of the career of the late Dr. Ralph Charles Brown.

Ralph Charles Brown, for so many years closely identified with the Alfred Hospital, Melbourne, died in Heidelberg House on March 12, 1947, in his seventy-seventh year. Born at St. Arnaud in 1870, the son of the Reverend Ralph Brown, his early boyhood was spent in the Mornington Peninsula, where his father occupied the pulpit in several churches. Educated at Toorak College, Brown then entered the University of Melbourne, where he became a student at Queen's College. After a most successful undergraduate career, in which he not only carried off a number of exhibitions and scholarships, but also represented his college in the tennis four, he graduated in 1892, passing head of the year with the scholarship in surgery and the Beaney Scholarship. His term as resident medical officer at the Melbourne Hospital was followed by one as acting medical superintendent. The testimonials given to Brown on his departure from the Melbourne Hospital in 1893, over such well-remembered names as Fitzgerald, Syme, Moore and Springthorpe, all tell of the high promise given by his abilities and personality. Through the good offices of a relative he was enabled to go abroad and he became a member and then a Fellow of the Royal College of Surgeons of England in 1895. Never very robust, Brown began to show signs of a pulmonary infection at this time, and actually sat for his final examination, in which he was successful, with a temperature of 102° F. With characteristic stubbornness and independence he decided that he would take up an appointment as a ship's surgeon to recover or to perish, rather than to return to his people as a burdensome invalid. Following several voyages to and around Africa, his health vastly improved, and on his return to Australia, with his financial resources at a very low ebb, Brown finally settled in Wycheproof in 1899. From the first day in practice there his success was assured, and within a year a debit had become a handsome credit belance and he had married Miss Marion Louisa Stead, of Melbourne. With further improvement in health Brown left the country for the city in 1903,



and for the next twenty-three years he lived and practised in Wellington Street, East St. Kilda, in close association with his friends, A. V. M. Anderson, R. H. Fetherston, J. P. Major and Roy Warne, who fully recognized his surgical abilities.

R. C. Brown's long association with the Alfred Hospital began as an honorary anæsthetist soon after his arrival in the city, and it was not until 1910 that he was appointed an out-patient surgeon. Two years later he was appointed indoors upon the retirement of H. M. O'Hara. The outbreak of World War I, in which he unsuccessfully offered his services, made heavy demands upon his time and strength, for to his public hospital and private work was added the time spent at the Repatriation Hospital at Caulfield, where he held appointment throughout the war years. This work was very dear to him and he devoted a great deal of time and thought to the problems of cranial defects and their repair. In 1926 Brown gratified a long-felt desire to renew his acquaintance with surgery overseas, and a year was spent in visiting the clinics of Europe and North America. Four years after his return he resigned from the active staff of the Alfred Hospital and was appointed honorary consulting surgeon.

Those who were in closest contact with R. C. Brown's work held it in the highest regard. Showmanship of any sort was entirely foreign to his nature. Slow, meticulously careful, gentle, thorough, perhaps he was at his best in his gastric surgery. In this his methods were akin to those of

Finsterer, of whose work he was a great admirer, and to whose results his own approximated. He was a Foundation Fellow of the Royal Australasian College of Surgeons, and in 1924 was elected a Fellow of the American College.

For many years "R.C." was an active member of the Medical Defence Association, of which he was vice-president. Here his talent for dispassionate survey of a situation found full scope. His hobbies, apart from an elaborate system for the orderly filing of papers, reports, reprints and cuttings from surgical papers and journals, were reading and gardening, in which he engaged at Frankston with his son and grandchildren until a few weeks before his death. R. C. Brown was a representative of a type of surgeon which is now becoming rare. Graduating as he did from a general practice in which he became known for his surgical leanings and ability, he remained throughout his life the physician who could operate skilfully and successfully.

### ALBERT EDWARD MARTIN.

WE are indebted to Dr. F. W. Simpson for the following account of the career of the late Dr. Albert Edward Martin.

Albert Edward Martin, M.D. (Dunelm.), F.R.C.S. (England), died on April 12, 1947. He was born at Ballarat, and practised as a chemist before studying medicine. After study at the University of London he qualified with the diplomas of Licenciate of the Royal College of Physicians and Member of the Royal College of Surgeons, and then went to the University of Durham, where he took the degree of doctor of medicine. He then returned to London, where he became a Fellow of the Royal College of Surgeons.

Martin practised at Plymouth and Ballarat before starting practice as an ophthalmologist at Perth. He was for nearly twenty years ophthalmic surgeon to the Perth Hospital, and was celebrated for his superb surgery. He was also for twenty-five years president of the School for Deaf and Dumb Children. He leaves a widow, a son (also a medical practitioner, Surgeon Lieutenant-Commander E. B. Martin) and four daughters. Western Australia has indeed been fortunate in having medical practitioners of the calibre of Albert Edward Martin to treat the sick and to bring to that State some of the culture and learning of England.

### WESTMORE FRANK STEPHENS.

WE regret to announce the death of Dr. Westmore Frank Stephens, which occurred on May 15, 1947, at Melbourne.

## Australian Medical Board Proceedings.

### NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Abbott, Lewsbe George, M.B., B.S., 1946 (Univ. Sydney), District Hospital, Wollongong.

Aird, Elizabeth, M.B., B.S., 1946 (Univ. Sydney), Rachel Forster Hospital, Redfern.

Anderson, Thomas William, M.B., B.S., 1946 (Univ. Sydney), Ryde District Soldiers' Memorial Hospital, Eastwood.

Booth, Howard Keith, M.B., B.S., 1946 (Univ. Sydney), Canterbury District Memorial Hospital, Canterbury.

Brown, Russell, M.B., B.S., 1946 (Univ. Sydney), Western Suburbs Hospital, Croydon.

Collings, Joseph Silver, M.B., B.S., 1946 (Univ. Sydney), Palmerston North Hospital, Palmerston North, New Zealand.

Creagh, Anthony, M.B., B.S., 1946 (Univ. Sydney), District Hospital, Goulburn.

Everingham, Douglas Nixon, M.B., B.S., 1946 (Univ. Sydney), Base Hospital, Rockhampton, Queensland.

The following additional qualifications have been registered:

Barrett, Alex Keith, 462, Marrickville Road, Marrickville (M.B., 1939, Univ. Sydney), B.S., 1946, Univ. Sydney.

Bors, John Kaspar, 9, Nyora Street, Killara (M.B., B.S., 1937, Univ. Sydney), F.R.C.S. (England), 1946.

Burton-Bradley, Burton Gyrth, 92, Ingham Avenue, Five Dock (M.B., 1945, Univ. Sydney), B.S., 1946, Univ. Sydney.

Bye, William Alick, 143, Macquarie Street, Sydney (M.B., Ch.M., 1923, Univ. Sydney), F.R.A.C.P., 1938.

Fleming, Justin Paul, 65, Muston Street, Mosman (M.B., B.S., 1940, Univ. Sydney), M.S., 1944, Univ. Sydney.

Fraser, Wallace Hugh, 217, Macquarie Street, Sydney (M.B., B.S., 1931, Univ. Sydney), D.P.M. (R.C.P. and S., England), 1937.

Gillies, Douglas Neil, 60, Long Street, South Strathfield (M.B., 1940, Univ. Sydney), B.S., 1946, Univ. Sydney.

### QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 to 1946, of Queensland, as duly qualified medical practitioners:

Clarke, Bruce Gibson, M.B., B.S., 1943 (Univ. Sydney), c.o. Dr. E. Mansfield, Cairns.

De Salis, Eric James Fane, M.B., 1942 (Univ. Sydney), Commonwealth Health Laboratory, Townsville.

Breitner, Lothar Franz, M.B., B.S., 1944 (Univ. Sydney), Repatriation General Hospital, Greenslopes, Brisbane.

Knight, Ronald Victor, M.B., B.S., 1945 (Univ. Sydney), General Hospital, Brisbane.

Everingham, Douglas Nixon, M.B., B.S., 1946 (Univ. Sydney), General Hospital, Rockhampton.

Butler, John Lachlan, M.B., B.S., 1943 (Univ. Sydney), Toowoomba Hospital, Toowoomba.

The following additional qualifications have been registered:

Sheehy, John Edward, Dalby, B.S., 1944 (Univ. Sydney).

Parry, Trevor Alexander, Alexandra, Wickham Terrace, Brisbane, M.R.C.P., 1946.

Fraser, Kenneth Barron, Ballow Chambers, Wickham Terrace, Brisbane, M.S., 1939 (Univ. Queensland), F.R.A.C.S., 1940.

## Medical Prizes.

### THE STAWELL PRIZE.

THE Stawell Prize, a memorial to Sir Richard Stawell, is open for competition. The amount of the prize is £30.

The conditions are:

1. The prize shall be awarded to the writer of the essay adjudged to be the best on a subject selected annually.
2. The subject for 1947 is "The Diagnosis and Treatment of the Anæmias".
3. The dissertation should be based on personal observation and experience of the writer.
4. The competition is open to graduates of any Australian university.
5. The trustees reserve the right to withhold the award.
6. Essays must be delivered to the Medical Secretary, British Medical Association (Victorian Branch), by 4 p.m. on December 31, 1947.
7. Each essay must be typewritten or printed and must not exceed 75,000 words in length.
8. Each essay must be distinguished by a motto and must be accompanied by a sealed envelope marked by the same motto, containing the name and address of the author.
9. The trustees reserve the right to publish the prize essay.

## Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Butler, John Lachlan, M.B., B.S., 1943 (Univ. Sydney), 62, Moruben Road, Mosman.

Finckh, Gertrude Dorothea, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Freeman, Jessie Strahorn, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Keller, Douglas Holcombe, M.B., B.S., 1946 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Kennedy, Kevin, M.B., 1946 (Univ. Sydney), 7, Queen Street, Randwick.

Lewis, David Herbert, M.B., B.S., 1927 (Univ. Sydney), Lawson, New South Wales.

McNamara, John William, M.B., B.S., 1936 (Univ. Sydney), 491, Military Road, Mosman.

Ormiston, Roslyn Isabel, M.B., B.S., 1946 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Percy, Neville, M.B., B.S., 1946 (Univ. Sydney), Sydney Hospital, Sydney.
Perrotet, Francis William, M.B., B.S., 1937 (Univ. Sydney), Flat 5, Greentrees, 11A, Silex Road, Mosman.
Renshaw, Maurice Edward, M.B., B.S., 1945 (Univ. Sydney), 308, High Street, Penrith.
Smith, Charles James Ross, M.B., B.S., 1946 (Univ. Sydney), 34, Fitzwilliam Road, Vaucluse.
Vautin, William Robert, M.B., B.S., 1946 (Univ. Sydney), Sydney Hospital, Sydney.

## Corrigendum.

AN error has appeared in the report of a reply by Dr. John Colebatch to a discussion on a case of congenital stenosis of the œsophagus, which was demonstrated at a meeting of the Melbourne Pædiatric Society and reported in the journal of May 10, 1947, at page 601. In reply the word "conservative" should replace the word "constructive". The sentence will then read: "It seemed from the natural history of the condition that conservative measures were worth a trial."

IN the article by E. R. Trethewie appearing in the issue of May 17, 1947, two errors have occurred. On page 613 the footnote to Table I should read "*c.t.*, milligrammes per cubic metre × minutes of exposure". On page 616 the figure ·00058 in the second column, fourteen lines from the bottom, should read 0·0058.

## Notice.

THE librarian of the Western Australian Branch is anxious to secure two copies of *The British Journal of Surgery* (January, 1920, and April, 1942) in order to complete two volumes. Any reader who is willing to give these journals to the Branch library is asked to communicate with the librarian at Shell House, St. George's Terrace, Perth.

## Medical Appointments.

Dr. N. A. Richards has been appointed gynæcological and orthopædic registrar at the Royal Adelaide Hospital, Adelaide.

Dr. A. J. Hakendorf has been appointed honorary dermatologist at the Mareeba Babies' Hospital, Adelaide.

The following have been appointed members of the Anti-Cancer Council of Victoria: Dr. Henry Searby, Dr. James Eric Clarke, Dr. Konrad Hiller, Dr. Leo Doyle, Dr. Balcombe Quick, Dr. Leslie Stuart Kidd, Dr. Charles Hugh Hembrow, Dr. Arthur Basil Corkill, Professor Frank Macfarlane Burnet, Dr. Clive Ernest Sandy, Dr. Cecil Ashley Marshall Renou, Dr. Douglas Thomas and Dr. Frank Gladstone Stephens.

Dr. Roy Gerard Fox has been appointed public vaccinator, Department of Health, Victoria.

Dr. W. Monz has been appointed visiting medical officer, in pursuance of the provisions of *The Aboriginals Preservation and Protection Acts*, 1939 to 1946, Aboriginal Settlement, Cherbourg, Queensland.

## Books Received.

"The 1946 Year Book of Pediatrics", edited by Isaac A. Abt, D.Sc., M.D., with the collaboration of Arthur F. Abt, M.D.; 1947. Chicago: The Year Book Publishers, Incorporated. 7″ × 4½″, pp. 464, with illustrations. Price: $3.75.

"Allergy in Practice", by Samuel M. Feinberg, M.D., with the collaboration of Oren C. Durham and Carl A. Dragstedt, Ph.D., M.D.; Second Edition; 1946. Chicago: The Year Book Publishers, Incorporated. 9″ × 6″, pp. 860, with illustrations.

"A Synopsis of Anæsthesia", by J. Alfred Lee, M.R.C.S., L.R.C.P., M.M.S.A., D.A.; 1947. Bristol: John Wright and Sons, Limited, London: Simpkin Marshall (1941), Limited. 7¼″ × 4½″, pp. 264, with illustrations. Price: 12s. 6d.

"The Rhesus Factor", by G. Fulton Roberts, M.A., M.B. (Cantab.); 1947. London: William Heinemann (Medical Books), Limited. 7¼″ × 4½″, pp. 50. Price: 3s. 6d.

"The Anatomy of the Nervous System: Its Development and Function", by Stephen Walter Ranson, M.D., Ph.D., revised by Sam Lillard Clark, M.D., Ph.D.; Eighth Edition; 1947. Philadelphia, London: W. B. Saunders, Company; Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10″ × 6½″, pp. 542, with many illustrations, some of them coloured. Price: 49s.

"The Australian Rhodes Review", Number 5; 1946. Published by the Association of Rhodes Scholars in Australia. 1946. Sydney, London: Angus and Robertson, Limited. 10¾″ × 7″, pp. 164. Price: 3s. 6d.

## Diary for the Month.

JUNE 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 4.—Victorian Branch, B.M.A.: Branch Meeting.
JUNE 4.—Western Australian Branch, B.M.A.: Council Meeting.
JUNE 5.—South Australian Branch, B.M.A.: Council Meeting.
JUNE 5.—New South Wales Branch, B.M.A.: Special Groups Committee.
JUNE 6.—Queensland Branch, B.M.A.: Branch Meeting.
JUNE 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

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